The Journal of Thoracic Surgery

VOL. 29 JANUARY, 1955 No. 1

Original Communications

FOUR YEARS' CLINICAL EXPERIENCE WITH INTERNAL MAMMARY ARTERY IMPLANTATION IN THE TREATMENT OF HUMAN CORONARY ARTERY INSUFFICIENCY INCLUDING ADDITIONAL EXPERIMENTAL STUDIES

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SINCE our first publication[1] in 1946 we have repeatedly reported experimental evidence suggesting that it was possible to graft a third artery into the left ventricle and have it function in coronary artery insufficiency. Since our last report[3] many points have been clarified, and the technique of implant has been greatly improved. It is now four years since our first human case of coronary artery insufficiency was operated upon.

A short review of previously published experimental evidence will be given, as well as recent information concerning blood flow studies made on the implanted internal mammary artery. The technique of implantation will be briefly outlined, and our results on the treatment of twenty-nine human cases of coronary artery insufficiency given.

At this juncture let us consider two important questions: (1) Just how necessary is surgery in the treatment of coronary artery insufficiency? (2) What experimental evidence is needed before a procedure can justifiably be used on human beings?

A review of published statistics concerning the longevity and disability of patients with coronary artery insufficiency is most enlightening and most definitely disturbing. There is no question that every physician knows of patients who have lived many years with angina pectoris. However, an analysis of the average longevity in a series of cases throws a different light on this question.

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Recent experimental data presented in this article were made possible by a Grant from the Department of Public Health & Welfare, Ottawa, Canada.

Read at the Thirty-fourth Annual Meeting of The American Association for Thoracic Surgery at Montreal, Quebec, May 3-5, 1954.

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In Table I, the longevity of patients suffering from angina pectoris is shown for a group of 5,027 cases reported in the recent literature. The average span of life in all cases is well under eight years and, as Parker and associates[11] and Bruck and associates,[12] who have reported the largest number of cases, point out, is probably five years. The figures quoted refer to angina pectoris, without consideration of the presence or absence of coronary thrombosis with infarction.

TABLE I. PROGNOSIS WITH ANGINA PECTORIS

AUTHOR	NUMBER OF PATIENTS WITH ANGINA PECTORIS	AVERAGE LONGEVITY (YEARS)
Herrick and Nuzun, 1918[4]	200	3.0
MacKenzie, 1923[5]	380	5.4 dead group
White, 1926[6]	200	3.4 dead group
White and Bland, 1931[7, 8]	500 followed 2 to 3 years: 445 dead; 52 alive	7.9 18.4
Eppinger and Levine, 1934[9]	141 fatal cases	4.5 average
Wedd and Smith, 1935[10]	166 fatal cases	5.8 average
Parker et al., 1946[11]	3,440 Mayo Clinic study	5.0 entire group
Bruck, Crumpacka, Dry, and Gage[12]	5,766 patients followed 5 to 23 years	15% dead end of first year: 9% dead each year thereafter. Average 5-year survival 58.4%. Entire group (normal pop. 86.9%). Average 10 years entire series 37.1% (normal pop. 70.4%)

In Table II is shown longevity after the first attack of coronary occlusion. The patients quoted by Conner and Holt,[13] and Levine and Rosenbaum,[15] may not have had the benefit of modern cardiological methods of diagnosis. This, however, cannot be said of those quoted by Katz and associates[17] (see Table III) and Sigler.[18] Although 81.5 per cent of Sigler's patients (females) and 83.1 per cent (males) were alive at the end of the second year, only 45.3 per cent of the males and 39.8 per cent of the females were alive at the end of the fifth year. These figures have been taken from a rather large source of 1,208 cases very carefully studied, and cannot be minimized. Katz and associates,[17] reporting 488 cases with infarction, show a much more serious picture. They report that 25 per cent of their patients were dead at the end of two months, 50 per cent at the end of one year, and 75 per cent at the end of three years.

TABLE II. LONGEVITY AFTER FIRST ATTACK OF CORONARY OCCLUSION

AUTHOR	NUMBER PATIENTS	SURVIVED FIRST ATTACK	FIRST YEAR	SECOND YEAR	FIFTH YEAR	TENTH YEAR
Conner and Holt, 1930[13]	287	117	75% well	56% well	21% well	
Levine and Rosenbaum, 1941[15]	372		75% alive	50% alive	25% alive	
Sigler, L. H., 1951[18]	1,208			81.5 (F) alive	39.8 (F) alive	10.4 (F)
				83.1 (M) alive	45.3 (M) alive	10.7 (M)

Average age onset for Sigler's group was 55 years.

In Table III is shown the longevity in a series of cases that have had coronary artery occlusion with infarction.

Now that we have discussed longevity in percentages, let us consider the ability of those who survive to work. There is no doubt that patients survive coronary artery occlusion with infarction; but what kind of lives do they lead? How many are capable of working and, if they are working, how many of them are symptom free? According to Friedberg[19] about 53 per cent return to work, but of these only 32 per cent are able to work full time. Thus, when

TABLE III. PROGNOSIS WITH CORONARY ARTERY OCCLUSION WITH INFARCTION

AUTHOR	NUMBER OF PATIENTS WITH CORONARY THROMBOSIS		MORTALITY
Conner and Holt, 1930[13]	287		16.2 dead—first attack
Bedford, 1935[14]	31		6 months
Levine and Rosenbaum, 1941[15]	372	101 fatal cases	3.4 years
Master et al., 1936[16]	243	8% dead up to time of discharge	
Katz et al., 1949[17]	488	with infarction	25% dead, 2 months 50% dead, end of 1 year 75% dead, end of 3 years
Sigler, L. H., 1949[18]	1,208	423 died after one attack or more	Av. longevity 3.8 yr. (M) Av. longevity 3.1 yr. (F)
		785 alive after one or more attacks	Av. longevity 4.9 yr. (M) Av. longevity 4.5 yr. (F)
Bruck and Crumpacka[12]	1,302	cases—previous infarction	5 yr. survival 45.9% 10 yr. survival 26.0%
Dry and Gage[12]	204	cases—subsequent infarction	5 yr. survival 41.2% 10 yr. survival 22.7%

we speak of approximately 50 per cent five-year survivals after coronary occlusion with infarction, one does not consider how many of these people who survived are useful citizens. In a general study of statistics one is surprised to find that only about 18 per cent of those who survive are able to return to a full and active life.

Now there is no doubt that every physician and every cardiologist knows of patients who have survived twenty years or longer following the onset of angina pectoris and who have worked and lived reasonably active lives. There are many such cases reported in the literature. These cases, however, are the exception rather than the rule, as every general practitioner knows.

Thus, it is clear that from the most optimistic figures, less than 50 per cent of patients with one coronary artery occlusion will be alive at the end of five years, and less than one-third of those who survive the first attack are asymptomatic and are capable of gainful occupation. It would seem from the figures presented that the disease progresses after the first coronary occlusion. Certainly this appears to be true between the first and fifth years after the initial attack (see Table II). Natural forces in the form of collaterals take over after the first coronary occlusion and maintain the myocardial muscle fibers in reasonable condition. Unfortunately, the process of coronary artery sclerosis is progressive, so that gradually more and more collaterals are shut

off and more and more muscle becomes fibrotic. Thus, if a new source of blood can be brought to the myocardium before it is destroyed, there is every reason to believe that further myocardial destruction can be stopped.

It must be remembered that the functional unit of the heart is the myocardial fiber. Once this is destroyed it cannot be replaced. When a great number of these fibers are destroyed, the reserve of the heart is greatly diminished and eventually a condition is reached where so many myocardial fibers have been destroyed and replaced by fibrous tissue that the most ideal method of revascularization can do nothing to help such a heart. Therefore, it is important to realize that the revascularization of the myocardium should be attempted as soon as possible to prevent progressive destruction and ultimate fatal loss of myocardial fibers. The onset of angina in severe form is, in our opinion, a definite indication for making an attempt at surgical revascularization of the myocardium. This will be discussed later under indications.

Experimental Criteria.—It has often been said of experimentation that what is true of the animal does not necessarily apply to man and therefore that experimental evidence is of little value. This is partially true. However, what is much more likely to be true is that if a procedure does not work in the heart of a dog or a pig, it is not at all likely to be of value in the heart of a human being. Far too many new medical and surgical treatments have been tried out on human beings without having been adequately tested in the laboratory.

Many new drugs have been used in the treatment of angina pectoris which were at first hailed with enthusiasm only to be discarded in the light of cold facts. Likewise, numerous surgical procedures have been devised, each to gain a measure of popularity largely dependent upon the personality and energy of the surgeon who developed the procedure, and each, in turn, like other forms of treatment, to be later abandoned. Practically all of these many therapeutic measures were founded on clinical and not upon laboratory data.

There are so many unknown factors in dealing with human coronary artery insufficiency that the value of any clinical method of treatment is most difficult to evaluate. Much time elapses and many lives are lost before the inadequacy of the treatment becomes apparent.

There is little question that before a method of surgical revascularization is attempted on human cases, certain fundamental experimental facts must be ascertained concerning that particular method. In our opinion, the minimal postulates that are necessary to prove the value of extracardiac circulation are five in number. These may be divided into (1) anatomical, and (2) functional; the latter has four subdivisions—a, b, c, and d.

1. *Anatomical.*—Proof must be shown that channels of arteriolar size or larger exist between the coronary circulation and the extracardiac source of blood. These anastomotic channels should be large and numerous enough to be capable of supplying the myocardium with as much blood as is normally supplied by the left coronary artery. These large anastomotic channels must be shown to be present for at least six months and preferably longer. The

anatomical proof of the existence of anastomotic channels between the coronary arterioles and an extra cardiac source of blood in itself is not sufficient; it is necessary to prove its functional value.

2. *Functional Value of an Extracardiac Blood Source.*—

a. *Survival following ligation of one of the major coronary vessels:* Survival of animals following ligation of one or more coronary vessels without infarction gives important functional evidence as to the value of an extracardiac source of blood in replacing an obstructed coronary vessel. This is true, provided all occluded arteries are inspected afterward to make certain that (1) the occlusion is complete, and (2) that there are no branches proximal to the site of occlusion which are open. The latter is particularly true of ligation of the anterior descending branch of the left coronary. Frequently the septal branch arises directly from the left coronary artery. Ligation of the anterior descending branch in such animals cannot be considered complete. It has been our custom, when this occurs, to exclude such animals from both the control and treated series. In this way a much clearer picture is obtained with regard to survival following anterior descending branch ligation.

b. *Interruption of the extracoronary circulation:* When animals survive anterior descending branch ligation following a revascularization procedure, it is assumed that the majority survive because of the blood supplied to the myocardium from an extracardiac source. If this is true, then interruption of this extracardiac source of blood should result in immediate death, or survival with a large infarction.

c. *Flow measurements of extracardiac blood source:* It is important to be able to measure the amount of blood delivered to the myocardium by an extracardiac blood source. In addition it should be shown that the blood which flows into the heart is actually used by the functional unit of the heart—the myocardial fiber. To determine whether fresh arterial blood to the heart does not by-pass the myocardium but is actually used by the heart muscle, oxygen consumption studies must be made.

d. *Production and treatment of artificially produced coronary artery insufficiency:* The production in animals of coronary artery insufficiency and its treatment by the introduction of an extracardiac source of blood is very important. This can be done by wrapping a piece of Dupont P-T 300 Cellophane around the origin of the anterior descending artery and slowly reducing, over several months, the exercise tolerance of the animal to a very low point. The successful introduction of an extracardiac source of blood into the ischemic myocardium should result in a return of the animal's tolerance to exercise as compared with the untreated and incapacitated animals.

Three different types of surgical procedure are now used in the treatment of human angina pectoris: (1) pericardial cardiopexy, (2) arterialization of the coronary sinus, and (3) internal mammary artery implant.

Before presenting the internal mammary artery method of revascularizing the ventricular myocardium, which has been developed and tested in the experi-

mental laboratories of McGill University during the past eight and one-half years, let us examine the anatomy of the myocardium and the pathology of coronary artery disease.

ARTERIALIZATION OF THE INTRAMURAL VESSELS OF THE LEFT VENTRICULAR MUSCLE BY MEANS OF INTERNAL MAMMARY ARTERY IMPLANTATION

Before proceeding to the details of this method for the revascularization of the ventricular muscle, a short review of the histological, anatomical, and pathological structure of the myocardium must be given. There are two factors now generally accepted upon which the procedure of internal mammary artery implantation is based: one is the anatomical pattern of the myocardial circulation, the other is the modern concept of the pathology of coronary artery sclerosis.

1. *Anatomical Structure of the Ventricular Myocardium.*—It is well known that there are many differences between cardiac and skeletal muscles. The differences in which we are most interested concern the peculiarly rich network of blood vessels which supply the myocardial muscle fibers. Actually, there exists within the myocardium a veritable sponge-work of vessels. Their arrangement is graphically shown in Fig. 1. As the smaller branches of the coronary arteries subdivide, they leave the surface of the heart and penetrate the myocardium at right angles. As they approach the endocardial surface they resume

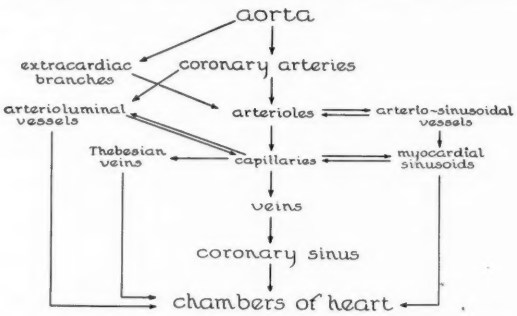

Fig. 1.—Relationship between the various components of the coronary circulation.

their horizontal direction and arborize into a rich arteriolar bed from which the capillaries arise. The arterioles also communicate directly with sinusoid arterial spaces which connect with the myocardial sinusoids. These latter vessels, with a lumen of from 50 to 250 μ in diameter, run a wandering course about the muscle fibers and anastomose with each other and the capillaries. Because of the structure of the myocardium, an implanted internal mammary artery with an open attached intercostal vessel fails to form a surrounding hematoma and remains patent for an indefinite period of time.

The second factor which indicates that internal mammary artery implant will be a successful method of treating coronary artery insufficiency is based on the pathologic character and distribution of coronary artery disease.

2. *Pathology of Coronary Artery Disease.*—The generally accepted idea that coronary artery disease is widely spread throughout the coronary tree has not

been borne out by pathologic study. Arteriosclerosis of the coronary arteries is usually, if not always, confined to the epicardial part of their courses and never involves the penetrating myocardial branches to any significant degree. Thus, in coronary artery sclerosis there is a vast network of arterioles, etc., deep within the myocardium which are free of disease.[21, 22] If arterial blood is delivered to this network, then it can reach all parts of the ventricular myocardium. An implanted internal mammary artery thus can bridge over the blocked superficial coronary vessels so that arterial blood can reach the intact deep arteriolar system of vessels which are still open and free of disease.

In the evolution of human coronary artery sclerosis a similar process occurs. It has been shown by Zoll[23] that in the normal human heart intercoronary anastomoses are rare. However, in the presence of arterial occlusion, intercoronary anastomoses develop distal to the point or points of occlusion. The development of such intercoronary anastomoses occurs frequently and probably prevents myocardial infarction when the occlusion develops slowly. Zoll[23] reports a study on a human heart with occlusion of the left anterior descending, left circumflex, and right coronary vessels, of such extent that the major portion of blood supplied to the heart came from a large separate conus artery which was connected with small branches below the points of occlusion in the descending coronary artery.

It is, therefore, not surprising to find that an implanted internal mammary artery carrying a high pressure and placed beyond the point of coronary artery occlusion and among the deep myocardial vessels into a low pressure area is capable of vascularizing the left ventricular vessels. We feel that the term "collateral circulation" should be reserved for intercoronary anastomoses. Revascularization of the myocardium by collateral circulation can only occur provided one of the major coronary vessels is still patent. In the presence of major disease of the coronary vessels this can only occur through mechanical development of an extracoronary source of blood. This occurs so rarely under natural conditions that it is useless to await its development. The futility of waiting is borne out by the high mortality rate of coronary disease treated by medical measures only.

The use of the left internal mammary artery as an extracoronary source of blood supply was first attempted by us experimentally in 1945 and, in 1950, we applied the procedure to our first human case of coronary artery insufficiency. The experimental data upon which the implant procedure is based have frequently been published but will be briefly reviewed here.

ANATOMICAL PROOF OF MAMMARY-CORONARY ANASTOMOSIS

Procedure.—The left internal mammary artery is freed from the chest wall from the fourth to the sixth intercostal space. The distal end is doubly ligated and transected between the ligatures. A tunnel is made in the anterior wall of the left ventricle and the freed portion of the artery is pulled into the tunnel and there fixed. The intercostal vessels which arise from the freed portion of the internal mammary artery are ligated, except for the sixth, which is transected just before the implant is pulled into the myocardial tunnel. The internal mammary artery is thus placed in a tunnel within the

ventricular myocardium, with an open, freely bleeding intercostal branch (Fig. 2). In another technique, the artery is buried with its end open (Figs. 6 and 7).

In previous publications[24-26] it was shown that an anastomosis developed between the implanted internal mammary artery and the left coronary circulation. The presence of such mammary-coronary anastomoses in dogs was proved in the following manner.

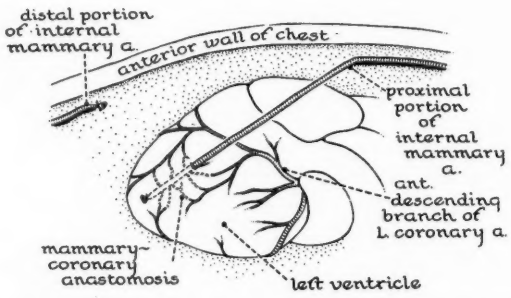
distal portion of internal mammary a.
anterior wall of chest
proximal portion of internal mammary a.
ant. descending branch of L. coronary a.
mammary-coronary anastomosis
left ventricle

Fig. 2.—Internal mammary artery detached from its chest wall attachment and buried in a tunnel in the left ventricular myocardium. Open stumps of intercostal not shown.

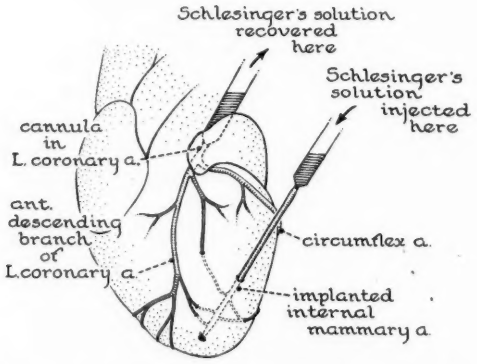
Schlesinger's solution recovered here
Schlesinger's solution injected here
cannula in L. coronary a.
ant. descending branch of L. coronary a.
circumflex a.
implanted internal mammary a.

Fig. 3.—The method by which Schlesinger's injection mass is introduced through a cannula placed in the internal mammary artery weeks to months after implantation. In the presence of coronary-mammary anastomosis, the injection mass spreads throughout the left coronary arterial network and emerges from a second cannula placed in the mouth of the left coronary artery.

Weeks or months after implantation the animals were sacrificed. Two separate cannulas were used: one was inserted into the internal mammary artery facing toward the heart, the other was tied in the mouth of the left coronary artery. The heart and attached internal mammary artery were removed from the thoracic cavity, and Schlesinger's injection mass was introduced through the internal mammary artery. This injection mass is known to pass freely through no vessel smaller than an arteriole 40 microns in diameter. Where there was a large anastomosis the injection mass was seen to enter the

internal mammary artery, spread throughout the coronary arteries of the left ventricle, and fill the cannula placed in the mouth of the left coronary artery (Fig. 3). This would indicate that the anastomosis is with the arterial rather than the venous side of the coronary circulation. After injection, x-rays were taken to show the distribution of the injection mass throughout the coronary circulation.

Following x-ray studies, sections were taken through the area of implantation and serial section studies were made. These serial sections showed branches leaving the implanted internal mammary artery, and these branches could be followed out into the myocardium. In Fig. 4, serial sections are shown which have been taken through the site of internal mammary artery implantation forty-eight days after implant. Note how the injection mass in the lumen of the implanted internal mammary artery can be followed out through a new branch into the myocardium.

Having proved the presence of mammary-coronary anastomosis by injection, by radiographic studies, and by serial section, it was then decided to attempt visualization of the anastomosis by plastic casts.

From a few days to many months after implantation, a plastic solution was injected into the proximal end of the internal mammary artery. At the same time, similar plastic solutions of different colors were introduced into the left coronary artery and the coronary veins. The injection mass did not enter the capillary vessels. Acid and alkaline solutions were used for digestion, and the resulting casts were studied under the dissecting microscope.

Two types of implants were studied, one in which the internal mammary artery was buried within the myocardium with its end open, and the other with it closed but with an attached open bleeding intercostal branch. When the internal mammary artery is buried with its end open and bleeding into a myocardial tunnel, an anastomosis occurs between it and the left coronary arterioles within five days. The entire left coronary arteriolar system can be filled with plastic substance through this implanted artery (Figs. 5 and 6). Such anastomoses have been found to be present at least two and one-half months after implantation (Fig. 7).

When the internal mammary artery is buried with its end closed, but with an open sixth intercostal artery, i.e., the standard implant described frequently during the past eight years, then the artery can be seen to branch at the end of twelve days (Fig. 8).

These branches grow and at the end of three to four weeks are well-formed arterioles which join up with arterioles lying in the left ventricular myocardium (Fig. 9). The anastomoses between the new branches from the internal mammary artery and the arterioles of the left coronary artery are, however, not usually fully developed under six weeks to two months.

In some of our injected specimens the casts show that the new branches join, not only with the coronary arterioles, but occasionally with the coronary veins (Fig. 7).

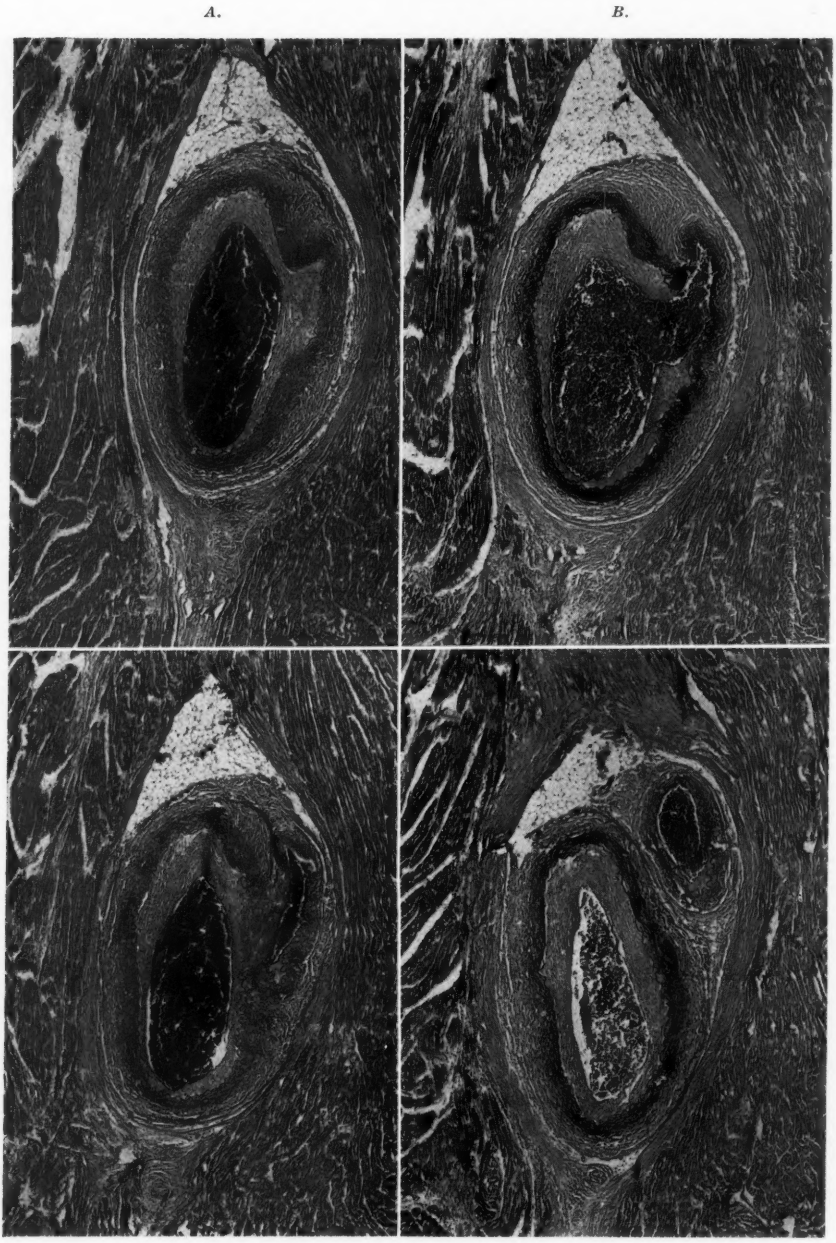

Fig. 4.—Serial sections through site of internal mammary artery implant forty-eight days after implantation showing branching of transplant, with injection in lumen.

FUNCTIONAL VALUE OF MAMMARY-CORONARY ANASTOMOSIS

Histoanatomical proof of anastomotic channels between extracardiac arterial sources and the coronary circulation gives no indication of its functional value. This is true not only of coronary-mammary anastomosis but of all other procedures used for augmenting myocardial circulation. Functional tests give the best indication of the value of the new arterial blood supply. These may be divided into four groups:

A. Survival experiments following ligation of the anterior descending branch of the left coronary artery.

B. Removal of extracoronary circulation.

C. Experimental production and treatment of coronary artery insufficiency.

D. Blood flow measurements of the internal mammary artery after implantation.

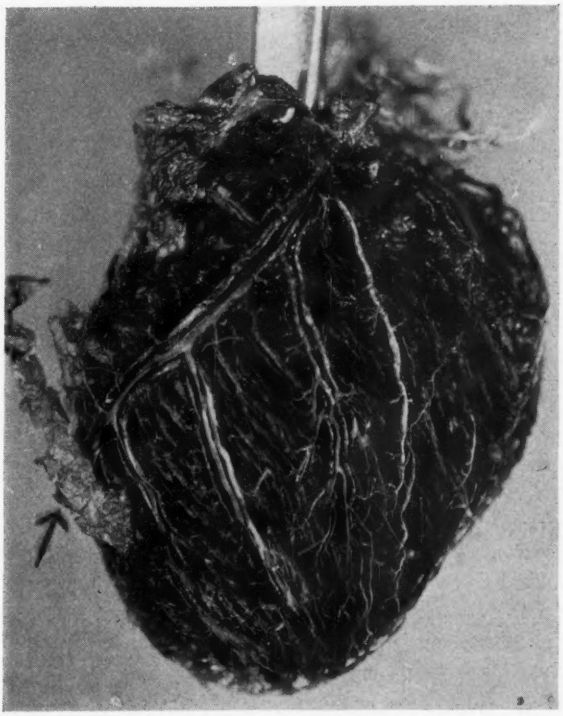

Fig. 5.—Internal mammary artery (arrow) freed from subclavian to sixth intercostal artery and open end implanted in 2-inch tunnel in left ventricular myocardium. Dog killed five days later in fight. Vinylite plastic injected through internal mammary artery is seen to fill arterial vessels of left ventricle.

A. *Survival Experiments Following Ligation of the Anterior Descending Branch of the Left Coronary Artery.*—There is some divergence of opinion in the literature as to the effects of ligation of the anterior descending branch of the

left coronary artery. Some claim a low mortality for this procedure, and others claim a high mortality. The majority of investigators have found the mortality in dogs to range from 70 to 80 per cent. In our series of ten control dogs, the average mortality rate from anterior descending branch ligation was 80 per cent. It is our belief that the great variation in mortality reported in the literature following anterior descending branch ligation is dependent upon the site of ligation.

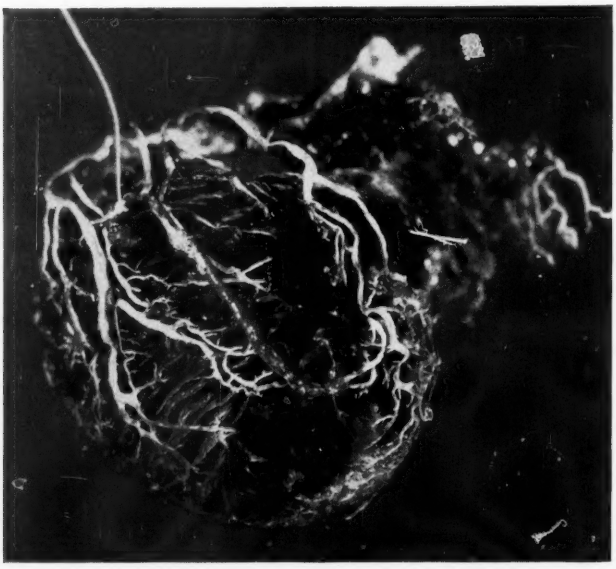

Fig. 6.—Same cast as Fig. 5, except that the coronary vessels superficial to the implanted internal mammary artery were removed to show underlying internal mammary artery. Note how in five days vessels leave the implanted internal mammary artery to join the left coronary arteries.

Method of ligation: The anterior descending branch of the left coronary artery should be ligated at its origin. The position of the ligature must be carefully checked by examining the left coronary artery to make certain that the ligature has included all branches, other than the circumflex. Frequently, one or two septal branches are missed, and occasionally branches arise from the main trunk of the left coronary artery.

An analysis of the results of anterior descending branch ligation is shown in Table IV. A total of 78 dogs had anterior descending branch ligation. There were 10 control animals, and 30 other animals in which the coronary circulation was not augmented. In this group of animals there was an overall 80 per cent mortality, with a 20 per cent survival with infarction. In 29 animals in which a large mammary-coronary anastomosis had developed, there were no deaths and no infarctions following anterior descending branch liga-

TABLE IV. EXPERIMENTAL DATA (VINEBERG AND ASSOCIATES)

	LIGATION ANTERIOR DESCENDING BRANCH OF THE LEFT CORONARY ARTERY			
	NO. OF DOGS	DEATH	SURVIVAL WITH INFARCTION	SURVIVAL WITHOUT INFARCTION
Control	10	80%	20%	0
Internal mammary artery implant and/or other experiments but without augmentation of the coronary circulation	39	80%	3%	17%
Internal mammary artery implant with coronary anastomosis	29			100%

Internal mammary artery after implantation forms anastomoses with coronary circulation in from 50 to 75 per cent of animals operated upon.

tion. Thus, it would seem that coronary-mammary anastomosis not only increases coronary circulation but gives protection against death and infarction.

The experimental data shown in Table V indicate that coronary-mammary anastomosis, when it occurs, is of value in protecting against death or infarction. However, because of the differences of opinion concerning the value of survival after descending branch ligation, a second group of functional experiments were performed.

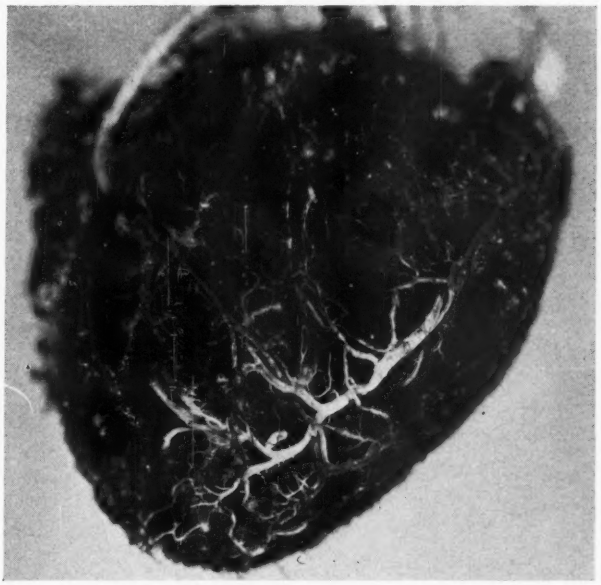

Fig. 7.—Internal mammary artery freed from chest wall from sixth intercostal artery to subclavian and open distal end buried in 2-inch tunnel in myocardium of left ventricle: partial ligation of origin anterior descending done simultaneously. Chest opened 77 days after operation and subclavian artery isolated and injected with India ink. The ink was seen to flow down the internal mammary artery into the heart, where it was seen in the coronary vessels. Note vast mammary-coronary artery anastomosis, also mammary-venous anastomosis (made by subsequently injecting plastic material into the internal mammary artery).

Fig. 8.—Internal mammary artery freed from chest wall between fourth and sixth intercostals and placed in 1-inch long tunnel in the ventricular myocardium with open stump of sixth intercostal. This animal died of pneumonia twelve days later. Cast made by injecting internal mammary artery with vinylite plastic, and cut away to show buried internal mammary artery with stump of sixth intercostal (arrow). Note tufting and branching from open stump of sixth intercostal attached to internal mammary artery.

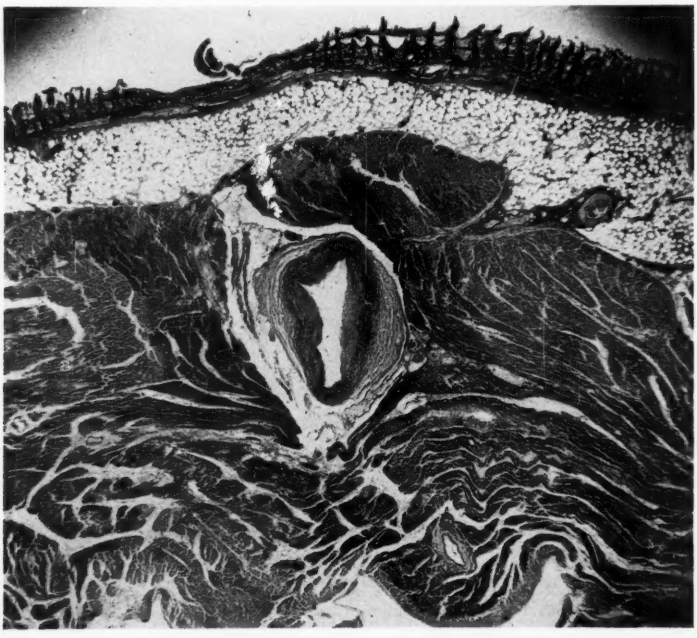

Fig. 9.—Section through human heart at site of internal mammary artery implant 62 hours later. Note how the vessel lies open between the myocardial fibers, and the absence of surrounding hematoma. This is remarkable because the artery was pulled into position with an attached open bleeding intercostal vessel. The absence of clot in the lumen of the vessel is likewise to be noted.

B. *Removal of Extracoronary Circulation.*—Animals which survived anterior descending branch ligation of the left coronary artery were subjected, four to five weeks later, to complete and sudden occlusion of the implanted internal mammary artery. If the internal mammary artery was maintaining the circulation of the left ventricle, then following ligation either death or infarction should result. This is exactly what happened. In three of the animals with an internal mammary artery implant which had survived anterior descending branch ligation, the internal mammary artery was ligated. One animal died immediately. Another animal died within twenty-four hours and displayed an edematous, cyanotic area of the anterior wall from a large infarct in the same location. Another animal survived, but examination of the sacrificed specimens revealed that multiple intercoronary anastomoses were present.

In all these animals there was a demonstrable mammary-coronary anastomosis. There can be little doubt that in those animals which died following internal mammary artery ligation, the circulation of the anterior portion of the left ventricle was being maintained by the internal mammary artery implant.

The evidence just presented suggested that the internal mammary artery after implantation into the left ventricular wall is capable of maintaining the left ventricular circulation. These experiments, however, were carried out on the hearts of normal animals. Therefore, it was necessary to attempt to artificially produce coronary artery insufficiency, and to treat it by internal mammary artery implantation. Thus, we set out to experimentally produce and treat coronary artery insufficiency and so establish a third functional test of the value of an internal mammary-coronary anastomosis.

C. *Experimental Production and Treatment of Coronary Artery Insufficiency.*—In this series of experiments coronary artery insufficiency was produced in dogs and treated by internal mammary artery implant. The first problem was to find a method of producing coronary artery occlusion which would simulate that which occurs in human coronary sclerosis. The second problem concerned the evaluation of coronary insufficiency when it was produced, and its response to treatment.

Method of producing coronary artery insufficiency: The anterior descending branch of the left coronary artery was exposed at its origin. Beneath the left auriculoventricular sulcus to the left of the pulmonary trunk, the epicardium overlying the vessel was incised and the bifurcation of the main coronary artery exposed. A doubly folded strip of Cellophane (Dupont-P-T 300), sterilized for twenty-four hours in a saturated solution of mercury oxycyanide, was twice wrapped completely, but loosely, around the origin of the anterior descending branch of the left coronary artery and fixed in position. Thus a segment of artery from 4 to 8 mm. long was completely surrounded with Cellophane.

The Cellophane initiated a fibroplastic reaction, with collagen deposition about the artery. As subsequent contracture of the new fibrous tissue

occurred, the lumen of the anterior descending branch of the left coronary artery was slowly narrowed. Gradually, that portion of the myocardium nourished by this vessel became ischemic.

At the end of the experiment when the animals were sacrificed, the anterior descending branch of the left coronary artery was probed to determine the extent of the narrowing. Sections were made through the artery at the site of the Cellophane wrap, and careful histologic studies made of the degree of coronary occlusion.

Method of evaluation of experimentally produced coronary insufficiency and its treatment. Exercise tolerance test: In human coronary artery insufficiency there are two outstanding symptoms, namely, substernal pain, and intolerance to exercise. In the dog, it is most difficult to detect the presence of pain. It is, however, not difficult to determine accurately the animal's tolerance or intolerance to exercise.

Clinical and pathologic experience have associated progressive exercise intolerance with progressive myocardial ischemia. Thus, in the experimental animal, progressive diminution of exercise tolerance has been accepted as evidence of myocardial ischemia. In this series of experiments, myocardial ischemia was produced by a Cellophane wrap placed around the origin of the anterior descending branch of the left coronary artery. Exercise tolerance studies were carried out before and after Cellophane wrap. Later, after internal mammary artery implant, they were repeated in order to establish whether improvement had occurred.

A treadmill was used to exercise the animals. This was made originally for man and was thus very satisfactory for exercising large animals. The exercise tolerance of each animal was established prior to wrapping the anterior descending branch with sclerosing type Cellophane. Two groups of animals were studied. The first group was composed of those animals in which coronary artery insufficiency was produced by Cellophane occlusion of the anterior descending branch, and treated by internal mammary artery implant. In this series the average running time was reduced from 9-12 minutes to 2.5-4 minutes, at 8½ miles per hour, three months after Cellophane wrap. These animals were given an internal mammary artery implant and, three to five months later, the running time had returned to 6 to 8 minutes, respectively, whereas the control animals had an average running time of 1.6 minutes at 8½ miles per hour. In those animals with an implant in which the running time had returned, there was a wide open coronary-mammary anastomosis.

In the second group of animals, an internal mammary artery implant was performed at the same time as the Cellophane wrap around the anterior descending branch. Where there was a widely patent mammary-coronary anastomosis, there was no appreciable reduction in exercise tolerance.

D. *Blood Flow Measurements of the Internal Mammary Artery After Implantation.*—Measurement of the amount of blood delivered to the heart by an implanted internal mammary artery, five, six, and seven months after im-

plantation have been made by means of a graduated biochemical burette, a stop watch, and a pressure tank (Fig. 10). The left subclavian and internal mammary arteries are isolated, the latter down to its point of entry into the heart, and a cannula placed in the subclavian artery. The outlet of the burette is connected with the cannula in the subclavian artery and the top of the burette to a pressure tank. The time taken for 10 c.c. of blood to flow

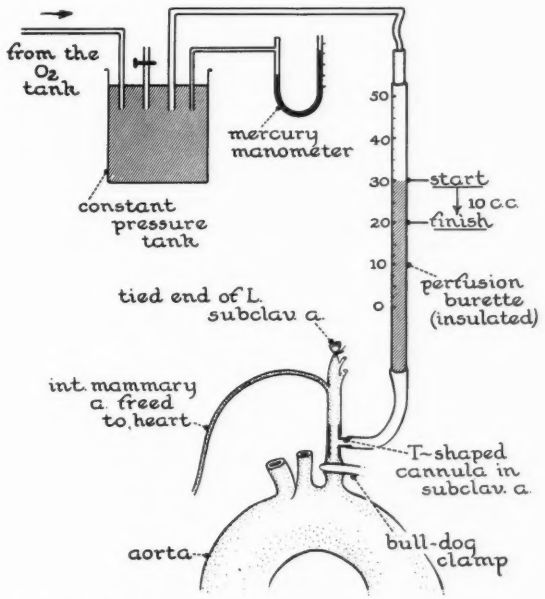

Fig. 10.—A diagrammatic representation of the perfusion apparatus for the measurement of blood flow through the implanted internal mammary artery. The rate of blood flow per minute is determined by clocking, with a stop watch, the time taken for 10 c.c. to flow through the internal mammary artery. Flow rates at pressures equal to, above, and below the carotid blood pressure were studied.

into the heart through the internal mammary artery was measured by a stop watch as expressed in cubic centimeters of flow per minute. The flow rate has been studied at perfusion pressures above, below, and equal to the carotid blood pressure.

The internal mammary artery is capable of delivering 85 c.c. of blood per minute (Table V), with both its ends and the sixth intercostal artery open. When the distal end of the internal mammary artery is tied and only the sixth intercostal is open, as it is when it is pulled into the myocardium, the perfusion rate is 45 c.c. per minute.

In four dogs studied five to seven months after implantation, and four to six months after ligation of the anterior descending branch, the flow rate varied from 3 c.c. to 21 c.c. when the perfusion pressure was equal to the carotid blood pressure, and from 14 c.c. to 51 c.c. when the perfusion pressure

was higher than the blood pressure. The blood flow through an implanted internal mammary artery into the heart five months after implantation is well shown in Table VI. The rate of 21 c.c. per minute when the blood and perfusion pressures are equal is indeed a functional amount of blood to be delivered to a heart via an implant. By increasing the perfusion pressure in relation to the blood pressure, the amount of blood sent into the heart via the internal mammary artery implant reached 51 c.c. per minute.

TABLE V. BLOOD FLOW STUDIES (INTERNAL MAMMARY ARTERY IMPLANTATION FOLLOWED BY LIGATION OF ANTERIOR DESCENDING BRANCH SIX WEEKS LATER)

ANIMAL NO.	CONTROL	TIME AFTER IMPLANT	CAROTID B/P (MM. HG)	SUBCLAVIAN ARTERY PERFUSION PRESSURE (MM. HG)	FLOW (C.C. PER/ MIN.)	RETROGRADE FLOW (C.C. PER/ MIN.)	ANASTOMOSIS
1	Internal mammary artery open end + open 6th intercostal		60	60	85		
	6th intercostal only		60	60	45		
128		7 months	0	120	11	Heart started to beat	Large
521		6 months	100	131	3		
			100	231	14		Small
			50	131	5	Slow	branches
			65	0		retrograde	
15		5 months	100	137	7		
			75	77	3		Large
			65	0		1.3	
33W		5 months	102	123	29		
			96	93	21		Large
			88	23		1	

Oxygen Saturation Studies.—After making the measurements just described, the animal was bled to death, the heart stopped, and a cannula was tied in the coronary sinus. Oxygen saturation measurements were made of the blood perfusing the internal mammary artery and recovered from the coronary sinus (Table VII). Table VII shows these figures and also demonstrates how the oxygen saturation dropped from 98 to 40 per cent after traversing the vessels of the heart. At this point the heart began to beat rhythmically again. It would seem that blood which goes through an implanted internal mammary artery enters the myocardial arteriolar system via mammary-coronary anastomoses and then enters the capillary or myocardial sinusoidal system where it yields its oxygen to the myocardial fibers and is returned in part via the venous and coronary sinus systems. In the experiment shown in Table VIII, sufficient oxygen was supplied to the myocardium through a 5-month-old internal mammary artery implant to an arrested heart to make it beat again.

The character of the mammary-coronary anastomosis five months after implantation and three and one-half months after ligation of the anterior

descending branch is shown in Fig. 11. Through these branches from 21 to 51 c.c. of blood per minute was delivered to the heart (Table VII). Sufficient oxygen was supplied to the myocardial fibers to start the heart beating again after its arrest.

TABLE VI. BLOOD FLOW THROUGH IMPLANTED INTERNAL MAMMARY ARTERY FIVE MONTHS AFTER IMPLANTATION AND FOUR MONTHS AFTER LIGATION OF ANTERIOR DESCENDING BRANCH

CAROTID ARTERY PRESSURE (MM. HG)	INTERNAL MAMMARY ARTERY PRESSURE AFTER CORRECTION FOR HEIGHT OF BLOOD COLUMN IN PERFUSION BURETTE	FLOW THROUGH IMPLANTED INTERNAL MAMMARY ARTERY INTO HEART (C.C./MIN.)
102	123	29
96	93	21
92	63	10
90	33	3
88	23	minus 1 (reverse flow)
Elevation Perfusion Pressure Above Carotid Blood Pressure		
86	123	32
86	153	43
82	183	51

(Dog 33W)

TABLE VII. MYOCARDIAL CONSUMPTION OF OXYGEN FROM BLOOD SUPPLIED THROUGH THE IMPLANTED INTERNAL MAMMARY ARTERY FIVE MONTHS AFTER IMPLANTATION

CAROTID PRESSURE (MM. HG) DOG KILLED BY BLEEDING HEART IN ARREST	INTERNAL MAMMARY ARTERY PERFUSION PRESSURE (MM. HG)	BLOOD FLOW THROUGH INT. MAMMARY IMPLANT (C.C./MIN.)	CORONARY SINUS FLOW (C.C./MIN.)	OXYGEN SATURATION		REMARKS
				INT. MAMM. BLOOD (PER CENT SATURATION)	CORONARY SINUS BLOOD (PER CENT SATURATION)	
0	123	54	30	98	60	
0	123			98	55	
0	123			98	55	
0	123			98	42	
0	123			98	40	Heart started to beat

DURATION AND CHARACTER OF CORONARY-MAMMARY ANASTOMOSIS

Glenn and his associates[27] have confirmed the development of an anastomosis between an implanted internal mammary artery and the coronary vessels. However, they stated that the new vessels tend to disappear at the end of eight weeks. They further suggested that the new vessels were small and resembled granulation tissue. In our series, the average interval from implant to sacrifice was eleven weeks (Table VIII). One animal was kept for fifty-eight weeks after implantation. The anastomoses at the end of that time not only persisted, but were large enough to protect against death and infarction following ligation of the anterior descending branch.

Not only has it been suggested that a coronary-mammary anastomosis tends to disappear at the end of eight weeks, but it has also been suggested that the anastomosis is capillary in nature. Serial sections of seven implants have been examined. Special preparations of these serial sections have been

made to show elastic and muscle tissue. The character of the new branches formed by the internal mammary artery implant has thus been studied. They have been found to be arterial in nature, containing both muscle and elastic tissue in their walls. These new arterial branches have been followed by serial section far out into the myocardium until they disappear.[27]

Fig. 11.—This is a cast of the heart of Dog 33W, flow measurement studies of which are shown in Table VI, and oxygen consumption studies in Table VII. This animal had an internal mammary artery implant five months before the cast and flow studies were made, and had survived anterior descending branch ligation three and one-half months previously.

FREQUENCY OF MAMMARY-CORONARY ANASTOMOSIS

We have heard the odd whisper that "the internal mammary artery implant is excellent when it works, but that most of the implanted arteries are blocked." In order to satisfy ourselves as well as others, we have reviewed

TABLE VIII. DURATION OF ANASTOMOSIS

TYPE OF ANASTOMOSIS	NO. OF ANIMALS	PRESENCE OF ANASTOMOSIS AFTER IMPLANT (WEEKS)	VALUE OF ANASTOMOSIS AFTER LIGATION OF ANTERIOR DESCENDING BRANCH OF THE LEFT CORONARY ARTERY
Microscopic Anastomosis	1	5	
	1	6	
	1	7	
Anastomosis small, due to thickened or partially thrombosed internal mammary artery	2	8	Protection against death or infarction afforded in three animals only
	2	11	
	2	12	
	2	13	
	1	14	
	12		
Macroscopic Anastomosis	1	6	
	2	7	
	5	9	No deaths or infarctions occurred
Anastomosis large	5	11	
	4	12	
	2	14	
	1	17	In coronary artery sclerosis, caused by Cellophane irritation, marked improvement in exercise tolerance occurred
Internal mammary artery patent	1	18	
	3	19	
	1	21	
	1	31	
	1	41	
	1	46	
	1	58	
	29		

TABLE IX. FREQUENCY OF MAMMARY-CORONARY ANASTOMOSIS STANDARD VINEBERG IMPLANT

	NO. DOGS	ANTERIOR DESCENDING BRANCH LIGATION	SURVIVED	WITHOUT INFARCT	ARTERY OPEN	ANASTOMOSIS
NORMAL HEART	9 (Vineberg)	Not done	–	–	4+	4
	6 (Niloff)	6	2	1	2	1
	11 (Miller)	11	3	7	7	8
	13 (Buller)	13	5	5	12	5
Total	39	30	10 (30%)	13	25 (65%)	18 (46%)
ISCHEMIC HEART	11	Partial ligation ant. desc. branch (23 gauge needle)	8 (72.7%)	5 (45.5%)	7 (63.6%)	6 (54.5%)
	5 Cellophane wrap ant. desc. branch followed by implant 3 to 4 months later		Exercise tolerance markedly reduced before I.M. implant. Returned nearly to normal in 2. Further reduced in one (abscess). Not tested in 2 (empyema)		4 (80%)	4 (80%) Condition of artery not recorded in 2
	5 Cellophane wrap ant. desc. branch — Implant int. mammary artery		2—Exercise tolerance reduced fr. 8-2½ min.		2 (100%)	2 (40%) small
			3—Exercise tolerance maintained at preop. levels		3 (100%)	3 (60%) large
Total	21				16 (76%)	15 (71%)

our experimental data concerning the frequency of mammary-coronary anastomoses in a series of sixty animals in which the standard internal mammary artery implant procedure was performed (Table IX).

Assuming that the proper technique is employed, the frequency of coronary-mammary anastomosis appears to be related to the heart's demand for fresh arterial blood. Thus, in thirty-nine animals receiving an internal mammary artery implant in normal hearts, there were eighteen (46 per cent) mammary-coronary anastomoses, and only 30 per cent of these protected against death from anterior descending branch ligation (according to our standards). Twenty-six (67 per cent) of the implanted arteries remained open many weeks to months later. When the hearts were made somewhat ischemic by partial ligation of the anterior descending branch, the anastomotic rate was 54 per cent, and the survival rate from anterior descending branch ligation was 72 per cent. Myocardial ischemia produced by wrapping Cellophane around the origin of the anterior descending branch simultaneously, or followed by internal mammary artery implant, resulted in an 80 to 100 per cent anastomotic rate. The average anastomotic rate in normal hearts was 46 per cent, with 67 per cent open vessels. In ischemic hearts the anastomotic rate averaged 71 per cent, with 86 per cent open vessels. This could be explained as follows:

The chance of an internal mammary artery implant branching will depend, theoretically, on its being placed in an area where the pressure is somewhat lower than within itself. The greater the differential pressure between the pressure within the implant and the pressure in the heart, the greater is the likelihood of the artery branching. Thus, when it is placed in normal heart muscle the pressure differentials are not as great as when it is placed in an ischemic heart muscle with a low pressure area. On this basis, the higher frequency of mammary-coronary anastomoses in ischemic hearts can be explained. It is likewise logical to deduct that patients suffering from coronary insufficiency should have about the same anastomotic rate as that seen in ischemic animal hearts.

TREATMENT OF HUMAN CORONARY ARTERY INSUFFICIENCY BY INTERNAL MAMMARY ARTERY IMPLANTATION

On the basis of the foregoing anatomical, functional and pathological facts, the internal mammary artery implant procedure has been used in the treatment of human cases of coronary artery insufficiency.

Selection of Patients.—The selection of patients for a new procedure is always difficult. We dislike classifications but, in a general way, we have found it convenient to divide our cases of coronary artery insufficiency into four groups:

Group 1. Mild or intermittent angina: In this group are those patients who experience an occasional attack of anginal pain on exercise, or during

an emotional disturbance. They are able to work and are capable of living reasonably normal lives. For this type of patient operation is not advisable—it is better to stop playing golf!

Group 2. Progressive coronary artery insufficiency without disability: Evidence of progressive coronary artery insufficiency is an important indication for surgery. Patients in this group may still be working, but there is evidence of progression of their disease. This may be shown in the electrocardiogram, or by an increase in the frequency of anginal attacks and, perhaps, progressive dyspnea.

Group 3. Partially or totally disabled, but without angina at rest: In this group are hundreds of thousands of people, men and women, who are unable to walk more than 50 to 100 feet, or to do 22 steps on the Master two-step test without anginal pain. Many suffer pain at rest, but in association with emotion, straining at stool, or a heavy meal. Some have night pain which awakens them. However, when sitting quietly in a chair, without stimuli, they are free of pain. Such cases have been accepted for surgery; in fact, surgery should be encouraged, as it has been shown that less than 50 per cent of such patients are alive at the end of five years. Another coronary artery occlusion may so lower their myocardial reserve as to place them in the hopeless, inoperable group of patients with status anginosus.

Group 4. Status anginosus: In this group are patients who experience angina at rest. These patients chew nitroglycerin tablets day and night. One of our patients in this group consumed 180 nitroglycerin tablets in two weeks. This type of patient should not be submitted to cardiac surgery. There is insufficient cardiac muscle left to revascularize. Generally, these patients are living on a pinpoint opening in the one artery which is still open. Such a small opening usually cannot supply sufficient blood to the myocardium to keep the patient alive during and after a major thoracic procedure. In addition, such patients generally have very little myocardial muscle left to revascularize.

Preoperative Investigation.—With each patient we have attempted to exclude all other possible causes for his symptoms. This has been done in spite of a well-established diagnosis of coronary artery insufficiency. Thus a search is made for intervertebral discs, infradiaphragmatic and supradiaphragmatic lesions and, in all cases, a psychiatric screening is done. A careful examination of serial electrocardiograms must be made for evidence of recent activity, as well as repeated sedimentation rates. In a few cases we have postponed operation for from four to six months on evidence of activity.

Contraindications.—(1) Evidence of left ventricular failure. The history of left ventricular failure during an attack of myocardial infarction, with recovery, is *not* a contraindication. However, the presence of left ventricular failure most definitely is a contraindication. (2) Enlargement of the left ventricle, unless it is due to hypertrophy associated with moderate hypertension. (3) Advanced cases of essential hypertension. (4) Evidence of recent myocardial infarction or of active disease. (5) Status angina.

PREOPERATIVE, OPERATIVE, AND POSTOPERATIVE CARE

Having determined that the patient has no contraindications to surgery, quinidine sulfate, 3 grains by mouth, is given twice on the preoperative day and once on the day of operation. An enema is given early in the afternoon of the preoperative day, as we have found that many patients are disturbed by it and have their sleep interrupted. Sodium luminal is administered, 3 grains, at 10 P.M., 11 P.M., and again at 6 A.M. Luminal sodium does not depress the blood pressure as other barbiturates do. At 7 A.M. on the day of operation, morphine or Demerol and 1/100 grain of atropine are given. It is very important that anesthesia be administered as soon as the patient arrives in the operating room. These patients generally are apprehensive and may develop a fresh coronary occlusion while awaiting operation. The surgeon must check to make certain that the patient is not suffering substernal pain just before the anesthetic is started. We failed to take notice of this warning signal in our first, sixth, and eighteenth patients. The first two patients died within sixty-two hours after operation from fresh coronary occlusions; the third developed pulmonary edema after the chest was opened and died eleven hours later.

Upon arrival in the operating room, the patient is given oxygen to breathe and, at the same time, a 2 per cent procaine hydrochloride drip is started. Induction is achieved with cyclopropane and anesthesia maintained with ether and oxygen. Once the patient is asleep a cut-down is started in the ankle: this is essential in case of a drop in blood pressure.

BLOOD PRESSURE MAINTENANCE

The maintenance of blood pressure is, we feel, extremely important. Neo-Synephrine drip is used when necessary and the blood pressure is maintained at a constant level throughout the operation. The desired blood pressure level is decided upon before operation and is based on the average blood pressure levels recorded four times daily during the ten days preceding operation. We have attempted to maintain blood pressure levels at not less than 20 mm. Hg below average preoperative levels. The coronary blood flow is dependent upon the diastolic blood pressure. Patients with coronary artery insufficiency are not like those patients with other types of heart disease in whom the coronary arterial system is comparatively healthy. Lowering of the diastolic pressure decreases the blood flow through the coronary vessels and lessens the amount of blood supplied to the myocardium. When this occurs in the presence of narrowed, calcified coronary arteries, we believe that coronary thrombosis is favored. In addition, the myocardium, which is already suffering from ischemia, receives insufficient blood for the maintenance of life, and the development of left ventricular failure and pulmonary edema are therefore favored.

FAILURE TO MAINTAIN BLOOD PRESSURE

The blood pressure in some of our cases has fallen with induction, and in others after the patient is postured on his right side. If the blood pressure

cannot be returned to proper levels with a slow Neo-Synephrine drip of 5 c.c. in 500 c.c. of 5 per cent glucose and water, then the operation is not begun. This has happened in two of our patients, neither of whom was operated upon.

OPERATIVE TECHNIQUE

A paper describing our operative technique in greater detail than is possible in this article is now being prepared.

The patient is placed in the right lateral position, and the fifth or sixth rib is removed, depending on the location of the apex. Procaine, 6 c.c. of 1 per cent in normal saline, is injected into the pericardium. The internal mammary artery is freed from the fourth to the sixth intercostal spaces inclusively. It is doubly ligated at its distal end and cut between ligatures. The mesial and diaphragmatic fat pads are cut away from the fibrous pericardium and left attached mesially and inferiorly, respectively. The fibrous pericardium is removed over the entire anterior surface of the heart and part of the inferior surface. The heart is inspected for areas of infarction and fibrosis, and the left coronary, anterior descending branch, and circumflex arteries are palpated for disease, which can be felt very readily.

A suture of No. 10 cotton is placed in the heart distal to the area selected for tunneling the myocardium. Proximal to this suture, a small incision is made in the epicardium. A tunnel of ¾ to 1 inch is made by blunt dissection, using a curved mosquito artery forceps. The cotton attached to the tied end of the proximal end of the severed internal mammary artery is picked up with the mosquito forceps and pulled through the tunnel. The sixth intercostal artery attached to the internal mammary artery is then cut and allowed to bleed freely. The internal mammary artery and bleeding sixth intercostal arteries are pulled into the myocardial tunnel. The end of the internal mammary artery is pulled out of the distal end of the tunnel where it is secured by the first suture placed in the heart.

Pericardial Fat Graft as Supplement to Internal Mammary Artery Implant.—During the past five years we have used various techniques of implantation hoping to increase the rate of mammary coronary anastomosis. One of these, first reported by Vineberg and Niloff, 1950,[26] was the pericardial fat wrap. At that time we thought the internal mammary artery tended to angulate less when surrounded by the pericardial fat. Subsequent experiments were not confirmatory. Bigelow* revived this idea, and pointed out the necessity of preserving the blood supply of the pericardial fat grafts. Since October, 1953, we have pulled the internal mammary artery through the mesial pericardial fat pad before implantation. In addition, we have used the pericardial fat pads as a supplement to the internal mammary artery in revascularizing the ventricular myocardium (Fig. 12) whenever possible. After the artery has been pulled through the mesial pericardial fat pad and implanted, areas of epicardium are removed on the anterolateral surface of the heart. The mesial fat pad is then sutured directly to the exposed myo-

*Personal communication.

cardial muscle. The inferior diaphragmatic fat pad is likewise sutured to a similar area on the posterior surface of the heart which has been denuded of its epicardium.*

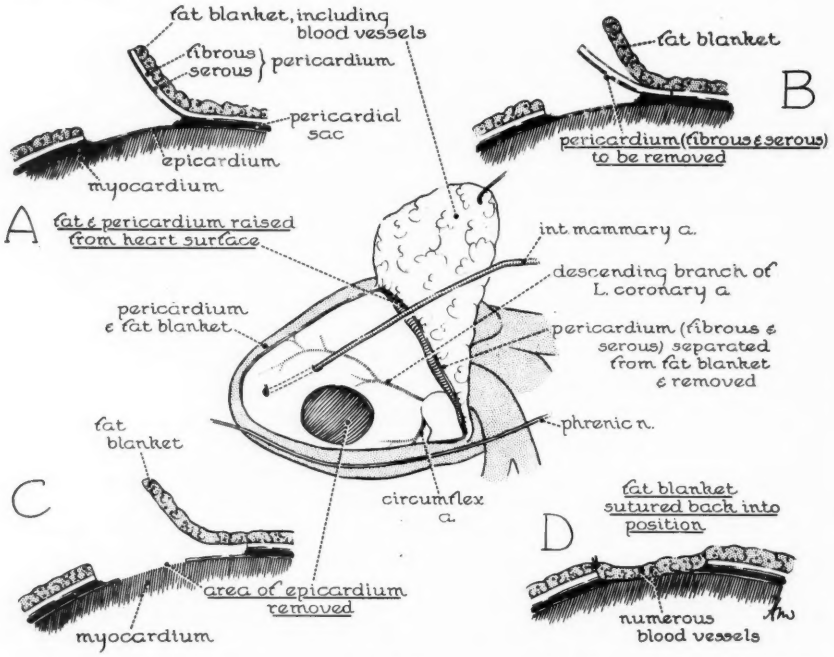

Fig. 12.—*A*, Pericardium composed of fat layer on top of a fibrous layer which is lined by parietal endothelium. Epicardium: a thin layer covering the surface of the heart which can be removed, exposing heart muscles; *B*, fat layer separated from fibrous pericardial layer; *C*, removal of fibrous pericardial layer and epicardium; *D*, placement of vascular fat blanket on denuded ventricle surface. Center: showing relationship of fat blanket to implanted internal mammary artery.

POSTOPERATIVE CARE

The two most important features of postoperative care are the maintenance of blood pressure at a constant level, and the maintenance of fluid and electrolyte balance. Neo-Synephrine continuous drip is used at 20 to 30 drops per minute depending on the weight of the patient, starting with 5 c.c. Neo-Synephrine in 500 c.c. of 5 per cent glucose and water. Sometimes this may have to be increased as much as 25 c.c. per 500 c.c. of 5 per cent glucose and water.

Patients are not moved for forty-eight hours. A movement of even 15 degrees can cause a marked drop in blood pressure. After forty-eight hours patients can be moved from the back to the right side, never to the left side. The amount of blood pressure instability seems to depend on the extent of

*For details of our first patient see preliminary report of the first human case operated upon using pericardial fat pads as a supplement to internal mammary artery implant, Canad. M. A. J. **70**: 76-78, 1954.

myocardial damage present before operation, as indicated by the exercise tolerance. Patients with low exercise tolerance invariably need blood pressure support for two, and sometimes five and six days. Others, with exercise tolerance of 20 to 29 steps, seem to require very little vasopressor help.

Neo-Synephrine aerosol is used every two hours as soon as the patient awakens from the anesthetic. Wangensteen suction is started early as some patients develop abdominal distention. This is discontinued as soon as there are abdominal cramps and passage of gas. Nasal oxygen is used routinely for twenty-four hours and longer if necessary. Quinidine sulfate is given by hypodermic, 3 grains twice daily. Usually, after the third day, Luminal sodium is substituted for Demerol. No hot or cold fluids are given, and the bedpan is never permitted. Mineral oil is started on the third day, as is Dicumarol. Usually patients are permitted out of bed on the sixteenth to the twenty-first day, and can usually leave for home around the fourth postoperative week.

RESULTS

This report deals with our experience with 29 patients who have undergone the internal mammary artery implant operation. This operation was completed in 28 patients. These cases have been divided into two groups—the first group of 10 patients followed for 1 to 4 years, and the second group of 19 patients followed from 2 to 11 months.

In the first group of 10 patients (Table X) all were disabled from 7 to 36 months, the average period of disability being 21 months. Half of these patients had had anterior and posterior infarctions, and only two patients showed no evidence of infarction. Exercise tolerance ranged from 50 to 200 feet. In this group followed one to four years after operation (Table XI), 3 patients died within 62 hours after operation; all 3 were cases of status angina. Seven survived the operation. Five, or 71 per cent, have returned to work and are working from 8 to 16 hours daily. One is working 16 hours daily and has slight pain on lifting or fast walking three years postoperatively. One worked for two years and then died pushing his car out of the snow; death was due to rupture of an old myocardial scar. Two still have angina postoperatively. One lived three years and died of aplastic anemia, the artery having pulled out of the heart. One is still alive and is showing improvement at the end of one year.

In the second group of 19 cases (Table XII) followed 1 to 10 months, there were only 3 patients in whom there was no apparent infarction. Two patients had anterior and posterior infarctions. Six were working at sedentary jobs, or with the aid of nitroglycerin. Thirteen were totally disabled from 2 to 21 months. There were 3 cases of angina status. Of these 19 patients, the operation was not completed in one, and there was one death (Table XIII). Seventeen patients underwent internal mammary implant and 10 of these had the supplementary procedure of pericardial fat pad graft.

TABLE X. FIRST TEN HUMAN CASES OF CORONARY ARTERY DISEASE FOLLOWED ONE TO FOUR YEARS

NAME	SEX	AGE	OCCUPATION	TIME DISABLED (MONTHS)	EXERCISE TOLERANCE	EATING	NIGHT	DYSPNEA	FATIGUE	PREVIOUS INFARCTION
				No Anginal Pain at Rest						
D. M.	M	54	Oil field worker	7	150 ft.	Yes	No	Yes	Yes	None
E. S.	M	49	Executive	24	100 ft.	Yes	No	Yes	No	Anteri poster
C. A.	M	55	Machinist welder	24	150 ft.	Yes	No	No	No	Large anteri
A. M.	M	55	Grocer	10	50 ft.	Yes	No	Yes	Yes	None
S. E.	M	57	Electrician	10	50 ft.	No	No	No	No	Large anteri
G. A.	M	52	Farmer	28	200 ft.	No	No	Yes	Marked	Poster
L. G. M.	M	39	Physician	24	50 ft.	Yes	Yes	Yes	Marked	Anteri poster
				Status Angina						
J. P.	M	53	Tailor	36	zero	Yes	Yes	No	No	Anteri poster
F. C.	M	57	Civil servant	36	100 ft.	Yes	Yes	Yes	No	Anteri poster
A. C.	M	46	Stevedore	12	150 ft.	Yes	Yes	Yes	No	Anteri poster

TABLE XI. FIRST TEN HUMAN CASES OF CORONARY ARTERY DISEASE FOLLOWED ONE TO FOUR YEARS

NAME	TIME SINCE OPERATION (MONTHS)	OCCUPATION	ANGINAL PAIN	EXERCISE TOLERANCE		ABILITY TO WORK	
				BEFORE	AFTER	BEFORE	AFTER
			No Anginal Pain at Rest				
D. M.	41	Oil field worker	None	150 ft.	Up to 10 miles	No	8 hr.
C. A.	40	Machinist	Following severe exercise only	150 ft.	½ mile	No	16 hr.
A. M.	34	Grocer	Slight	50 ft.	¼ mile	No	Works, and rides horseback
G. A.	16	Farmer	None	200 ft.	Ploughed 1,600 acres	No	Working
L. G. M.	14	Physician	Yes	50 ft.	Was 1 mile. Influenza and extrasystoles limited	No	No
			Angina Status				
			Three patients died in first 62 hours postoperatively				
			Died Recently				
E. S.*	37	Executive	Yes	100 ft.	100 ft. or less	No	No
S. E.†	22	Electrician	Occasional	50 ft.	½ mile	No	8 hr.

*E. S. One failure in group. Died of aplastic anemia—artery blocked.

†S. E. Was returning from work: pushed car out of snow and died of rupture of old scar, interventricular septum.

TABLE XII. SECOND GROUP OF 19 CASES OF CORONARY ARTERY DISEASE FOLLOWED 1 TO 10 MONTHS

NAME	SEX	AGE	OCCUPATION	TIME DISABLED (MONTHS)	EXERCISE TOLERANCE	EATING	NIGHT	DYSPNEA	FATIGUE	PREVIOUS INFARCTION
					No Anginal Pain at Rest					
C. H.	M	45	Engineer	17	11 steps	Yes	Yes	Yes	Yes	Posterior
M. B.	M	36	RR sec. man	18	30 steps	Yes	Yes	Yes	Yes	Ant. septal
W. H.	M	53	Sta. engineer	21	30 steps	No	Yes	Yes	Yes	Posterior
B. P.	M	52	Cook	9	29 steps	Yes	No	No	No	Posterior
P. O.	M	49	Carman	Working, 10 nitros.	16 steps	No	No	No	No	Posterior
C. H.	M	48	Accountant	Working	16 steps	No	No	Yes	No	Operation not completed
G. T.	M	36	Air pilot	6	20 steps	Yes	No	No	Yes	Posterior
W. B.	M	53	Salesman	Working	49 steps	No	No	No	Yes	Posterior
L. Th.	M	43	Oilman	12	11 steps	Yes	Yes	Yes	Yes	Post. wall
J. H.	M	40	Metal worker	17	16 steps	No	No	Yes	Yes	Large anterior
C. L.	M	53	Tractor rep.	12	15 steps	No	No	Yes	Yes	Ant. posterior
R. R.	M	58	Mechanic	3	19 steps	No	No	No	Yes	Posterior
				sedentary	No pain					
C. T.	M	54	Foreman	17	½ block	No	No	Yes	Yes	Posterior
F. T.	M	59	Merchant	10	1 block	No	No	No	Yes	Posterior
C. S.	M	52	Carpenter	3 years sedentary	25 steps	Yes	No	No	Yes	Posterior
A. S.	M	55	Salesman	Working	1 block	No	No	No	Yes	None
					Angina Status					
T. K.	M	55	Hotel man	2 months	Too sick	Yes	Yes	No	Yes	None. Vessels sclerosed
C. A.	M	60	Aircraft	6 months	Too sick	Yes	Yes	Yes	Yes	Apical. Ant. lateral
PAR.	M	31	Vet. amputee	9 months	Too sick	Yes	Yes	Yes	Yes	Posterior

TABLE XIII. RESULTS. SECOND GROUP 19 CASES FOLLOWED 1 TO 10 MONTHS

NAME	TIME SINCE OP. (MONTHS)	OCCUPATION	ANGINAL PAIN	EXERCISE TOLERANCE		ABILITY TO WORK	
				BEFORE	AFTER	BEFORE	AFTER
C. Hil	11	Engineer	Yes	11 steps	Not tested, improved	No	Not working
M. B.	9	RR sec. man	Occasional	30 steps	½ mile	No	Part time
W. Hart	6	Sta. eng.	No	30 steps	3-4 miles	No	Working
B. Par	6	Cook	Yes	29 steps	Few blocks	No	No
P. Ol	4	Carman	Occasional	16 steps	Improved, has reduced nitros. from 10 to 0	Yes	Full day
G. T.	5	Air pilot	No	20 steps	1-2 miles	No	Full day
W. B.	4½	Salesman	No	49 steps	Not known	Yes	Part time
L. Th.	4	Oilman	No	11 steps	Not known	No	Part time
J. Han	4	Metal worker	No	14 steps	1 mile	No	Full time
C. Iss	4	Tractor rep.	No	15 steps	Marked improv.	No	Full day
R. Ran.	4	Mechanic	No	19 steps	Complication: empyema, still draining.		
C. Tol	4	Foreman	No	½ block	Unknown	No	Working
F. Trev	2	Merchant	No	1 block	25 yd.	Seden.	Working
C. Shep.	1	Carpenter	No	25 steps	Not known	Seden.	Part time
				Angina Status			
T. Kel	5	Hotel man	?	None	Not known	No	Not known
C. Adams	5	Aircraft	Yes	None	1 mile	No	Working
Par	5	Vet. Symes amp.	Yes	None	50% improved	No	Walking
A. Ste	55	Salesman. Died on operating table, pulmonary edema. One death in 19 cases.					

Twelve, or 67 per cent, are free of pain, with increased exercise tolerance. Three still have pain but are improved. One has severe pain with no improvement. One has not been followed. Thus, of the 29 patients there were:

Twenty-three patients without angina at rest. In this group there was one death, or a mortality rate of 4.1 per cent.

Of the 6 patients with angina status, 3 died in the early series, giving a 50 per cent mortality.

The mortality rate for the entire group is 14 per cent.

There were 24 patients who survived, 23 of whom had the implant operation. Fifteen, or 60 per cent, are now completely free of pain. Seventeen, or 73 per cent, have returned to work after operation. Many are working 8 to 16 hours daily at hard physical labor.

SUMMARY

The clinical results of internal mammary artery implantation in the treatment of coronary artery insufficiency are most encouraging.

About 70 per cent of our patients are markedly improved and have returned to work.

For cases without angina at rest the mortality rate is low—4.3 per cent.

In the experimental animal, the implanted internal mammary artery has been shown to send out large branches in the ventricular myocardium in 71 per cent. These branches are large enough to carry a supply of fresh blood into the heart at a rate as high as 51 c.c. per minute.

The pericardial fat pads are used as grafts applied to the surface of the left ventricle to supplement the internal mammary artery revascularization of the left ventricle.

The results on our human patients are beginning to parallel very closely the frequency of mammary-coronary anastomosis demonstrated in our animal experiments.

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Discussion

DR. W. B. NEPTUNE, Boston, Mass.—In the Overholt group we have been interested in the problem also and in the past have done several of these procedures on clinical cases. However, I should like to present some of my own observations.

(Slide) Of 18 experimental procedures done on normal animals, 2 were found to be thrombosed, 5 showed no anastomosis, 5 supplied a small capillary bed, 6 went to the coronary system and, of these, 3 went to the arteries and three to the veins. If we make a section through the transplanted artery, note the intense intimal proliferation with decreased cross-diameter of the lumen. This is nine months after surgery and I think Dr. Vineberg's sections showed similar findings. This has been uniform in our experiments.

Our anatomic studies are based upon injections in living animals, using a 4 per cent gelatin solution, containing a colloidal suspension of carbon particles, 27 mμ in diameter, which has been suitable for the demonstration of myocardial capillary bed. This has been injected through the internal mammary artery at constant body temperature and at the same pressure as found in the arch of the aorta. For the sake of this discussion we will forget all cases except the most ideal, and those are the only ones I wish to speak of.

Grossly, we have found that the carbon particles are limited to the anterior surface of the heart and around the myocardial tunnel itself. If we make a tangential section through this area we note that the carbon particles again are right around the myocardial tunnel in Zone I and in Zone II (20 mm. from the tunnel) we do not see much. This is a low-power field of the entire section of the heart showing the transplanted artery. If we

make a high-power section through Zone I, right around the tunnel itself, we see an excellent demonstration of the myocardial capillary bed; excellent filling, no question about it. In Zone II, which is 20 mm. away, I do not think we can demonstrate any evidence of myocardial capillary filling. If we make a cleared section of this same area after the Spalteholtz technique, we cannot demonstrate any evidence of vascular pattern.

Just to let you know that we can demonstrate capillary filling if we make a cleared section of a normal heart injected through the coronary artery, this is the normal pattern which we can demonstrate. These studies to us would suggest that revascularization by means of a transplanted artery in a myocardial tunnel, per se, offers little in the way of flow or area supplied.

DR. A. R. HENDERSON, Asbury Park, N. J.—Much impressed with the results reported by Drs. Vineberg, Samuel Thompson, Beck, and others during the past few years, we have repeated the various methods designed to increase collateral flow of blood to the heart in the experimental laboratory. Although a considerable portion of the dog's heart could be vascularized by any of the procedures advocated (e.g., pericardial insufflation with talc, internal mammary artery implantation, arterialization of the coronary sinus), we were very much disgusted with the unreliability of any one procedure to function as a revascularization phenomenon. Since coronary artery disease usually involves the entire coronary system, it was felt that in the surgical treatment of human cases no one procedure should be relied upon which would most likely vascularize only a portion of the failing coronary circulation.

For that reason, in treating patients surgically, we have subjected them to combinations of revascularization procedures. Thus, in one operation, 20 patients have had internal mammary implantations, pericardial poudrage, and left upper dorsal sympathectomy. Sympathectomy (T_1-T_4) was performed not to relieve the patient of angina but because we have been very much impressed with recent work on the role af sympathetic secretory mechanisms (particularly the oversecretion of norepinephrine), in producing an oxygen-wasting type of myocardial metabolism. Denervation, in these cases, probably reduces the oxygen requirements of the myocardium, thus reducing demands upon the coronary flow.

Of the 20 patients thus far operated upon within the past fourteen months, only one has died. There have been no operative deaths. The one patient succumbed to a massive occlusion eight days postoperatively. It is hardly fair to judge the results of such combined therapy at this time, but thus far the clinical results are gratifying. Dr. Vineberg is certainly to be congratulated for his persistent efforts and his contribution to the surgical management of coronary artery disease.

DR. A. D. CRECCA, Newark, N. J.—We were first introduced to this surgery of coronary artery disease in 1951. At that time our mentors were Drs. Bailey and Beck. We started with the Beck procedure, that is, the Beck No. 2 which, as you know, is a shunt operation, and we had some very interesting observations on that and on the other operations. We have used all types to date in 26 patients: the Beck No. 2, the Beck No. 1, the Thompson operation, and also the Vineberg. Clinically, those patients who survived all seemed to benefit. An observation on the Beck procedure, I think, is of importance to this group. One of our patients after two years developed severe edema, and cardiac catheterization showed that his shunt was now pouring 39 per cent of the blood volume through the shunt into his right auricle. The edema was uncontrollable by medical means, and we submitted him to operation in spite of the severe edema, and tied off the coronary sinus at its ostium completely. We feel that the coronary venous system had thrombosed and, after this, had allowed the shunt circulation to pour into the right auricle. After this tying-off procedure the edema completely disappeared within 48 hours. This patient, who was president of our "Cardiac Surgery Patients Club," is well and very active in our program to educate patients who need surgery.

Our over-all mortality is 11 per cent. Of the eight who had Beck No. 2 procedures, two patients died; one died we feel from Dicumarol intoxication or overdosage, because

after the eighth day the aorta blew out and at autopsy there was absolutely no evidence of healing. The second patient died of cardiac arrest several hours after operation. A massage procedure was undertaken for three hours, the patient had no evidence of neurologic damage, the heart returned to function for no longer than 60 to 80 seconds at a time under massage and stimulation and, finally, after three hours we decided that the game was up.

We had two Vineberg operations with no mortality; three Thompson procedures with one death. We use the Thompson operation on cases with status angina, and feel that the procedure can be done more swiftly without much stress on the patient and is well tolerated.

DR. ALFRED GOLDMAN, Beverly Hills, Calif.—About a year and a half ago we developed the concept that perhaps some beta radiation might affect the ischemic heart. This concept was based upon the observations that radon ointments and ointments made with P-32 are capable of stimulating capillary ingrowth in chronic ulcerations associated with arteriosclerosis. The problem was to test this in dogs, and I would like to present some preliminary observations.

We used approximately 83 dogs. The largest branch of the anterior descending artery was ligated and at the same time 3 mc. of colloidal radioactive chromic phosphate was instilled into the pericardium. After similar anterior coronary ligation in the control animals the same volume 6 c.c. of nonradioactive colloidal phosphate was instilled in the pericardium and the pericardium was closed tightly. The complete results of this procedure I cannot give at this time except to say that the dogs survived this procedure; grossly, scarred infarcts which were vascular at the time of sacrifice were observed. We still have some 30 animals in this group to sacrifice, and this is a preliminary report.

(Slide) This is the section of the myocardium from a dog that was sacrificed at 64 days, and you will see the large infarct with ingrowth of capillaries and small vessels. This is the autoradiograph which shows the activity of the radioactive phosphate tending to localize on the epicardial surface and over the area of infarction.

(Slide) This is a microphotograph of the same animal showing vascularization in the infarcted area.

I simply present this as a different method, perhaps a different approach to the problem of revascularization of the ischemic heart.

DR. IVAN D. BARONOFSKY, Minneapolis, Minn.—I should like to thank you for the privilege of the floor. Dr. Vineberg is to be congratulated on his ideas and results. Yesterday we heard Dr. Key present a paper on the use of a jejunal transplant to the heart in order to revascularize the heart. Independently, my resident, Dr. Joseph Sprafka, and I, at the Ancker Hospital in St. Paul, have been using a somewhat similar procedure. We have used a defunctionated Roux-en-Y loop of jejunum or ileum and attached it to the heart. We have also used a flap of stomach muscularis.

(Slide) This shows the Roux-en-Y loop of intestine brought up to the diaphragm. The serosa of the defunctionated loop is stripped off for a good distance. This is very easily accomplished.

(Slide) Here is the loop being attached to the heart. The epicardium has been stripped off the heart using a very easy maneuver. The gut must be placed over the branches of the anterior descending coronary artery.

(Slide) Here is an injected specimen showing the gut attached to the heart. The mesenteric artery has been injected and, as you see, the entire coronary system has been visualized by the injection of the mesenteric vessel.

(Slide) Here is another injected specimen in which the anterior descending artery has been cut off and, as you can see, there is an excellent filling of the coronary system by the injection of the mesenteric artery.

We have done one human case recently, and it is much too early to arrive at any conclusion.

DR. SAMUEL A. THOMPSON, New York, N. Y.—There are two general principles involved in myocardial revascularization; first the use of some form of extracardiac anastomosis and, second, the stimulation and further development of the residual myocardial circulation. Dr. Vineberg has very beautifully demonstrated the use of an extracardiac anastomosis. We do not feel that the potentialities of the residual myocardial circulation have been sufficiently emphasized. This residual myocardial circulation comes partly from the coronary system and partly from small vessels around the pulmonary artery, around the orifices of the superior and inferior venae cavae and from the pericardium and the aorta at the base of the heart.

This residual circulation is able to furnish a tremendous collateral supply under proper stimulation, and it may be true that the greatest good effect of all the operative procedures comes from this source of supply rather than from the extracardiac source. For sixteen years we have been using a simple but very effective method in the treatment of coronary insufficiency, which utilizes the stimulation of the residual myocardial circulation plus the advantage of an extracardiac anastomosis and tissue graft. We have done the operation in 134 cases. The operation is known as cardiopexy, sometimes called "The Talcum Powder Heart Operation," and occasionally I have been asked "What fragrance do you prefer?"

The operation is done through a small anterior incision over the fifth cartilage, the pericardial sac is opened and all the pericardial fluid is removed. The surface of the heart is completely covered with magnesium silicate powder, some 2 to 6 teaspoons by volume and 4 to 12 Gm. by weight. We do not believe it is necessary to scarify or attempt to remove the epicardium as the silicate powder is a very strong irritant, and produces a violent inflammatory reaction. This reaction is sustained in some form for the lifetime of that patient. The reaction has a recognizable pattern of behavior and a predictable end result. The simplicity of the operation with the low mortality has much to recommend it. We believe the end results are comparable to any method used.

We have approximately 10 per cent of poor results. Poor results mean anything less than 50 per cent improvement. The other 90 per cent of patients are improved more than 50 per cent. We have 40 per cent who are more or less normal. Normal means no medication and no restriction in physical activity.

DR. W. G. BIGELOW, Toronto, Ontario.—Thank you for the privilege of discussing Dr. Vineberg's paper. All who have studied this problem are well aware of the difficulties of assessing benefits from any attempt to revascularize the heart both in the experimental animal and in human cases. However, because of Dr. Vineberg's fine experimental results we were tempted to investigate the problem in our own laboratory. We have not seriously studied the apparent protection this operation affords to coronary ligation but, rather, have turned our attention to a study of the fate of the implanted artery. We examined, rather carefully, three internal mammary transplants in animals nine months after their insertion and after anterior descending branch ligation. It was the injestion results and the pathologic picture in which we were interested.

(Slide) We got good injection studies in each. The dye entered the coronary artery and not the vein in all three.

(Slide) What we were interested in was the principle which Dr. Vineberg was establishing. It did not seem credible to us that one could put an artery in an organ and have that artery branch out and revascularize the organ. Following Dr. Vineberg's technique, the intercostal branches were cut and allowed to bleed vigorously just before the artery was drawn into the tunnel.

After overcoming some initial skepticism on the part of the pathologists, serial sections were studied from the whole area of implantation. What we found was interesting. From the ends of the open intercostals in each case there were invariably one to three newly formed branches of reasonable size. It is impossible to assess their exact size pathologically. The new vessel was identified by the absence of an internal elastic lamina. The open intercostals proved the only apparent source of new vessels. In this particular case the artery had been thrombosed and recanalized.

A similar pathologic picture and a similar result from injection has been obtained in each animal. Dr. Ritchie has pointed out that the vulnerable area for the implanted area is where it enters the heart at the level of the epicardium. In recent studies of coronary artery disease, that has been proved to be the area where sclerotic changes are most likely to occur. The intramural section of the artery is usually well protected and shows little change. I am afraid that this area may show progressive narrowing over a longer period and be exposed to the danger of thrombosis.

It is a very interesting fundamental phenomenon: the concept of putting a major artery in an organ and having it supply the organ with branches of that size. It warrants more study.

DR. CHARLES B. RIPSTEIN, Brooklyn, N. Y.—I think Dr. Vineberg is to be commended on his excellent experimental work, but I would like to interject a word of caution on the wide application of any of these procedures to human material. In 1948, I published a review of all the surgical procedures then being used for coronary artery disease and came to the rather reluctant conclusion that none of them really offered any lasting benefit. I am presently completing another such review and am again, reluctantly, of course, coming to the same conclusion.

DR. ARTHUR M. VINEBERG (Closing).—The results reported by Dr. Neptune on a small series of animals subjected to internal mammary artery implant are not surprising. They are exactly the same type of results that my resident or research Fellow obtains in the first few months when working with us. For example, this year Dr. William Buller, who started to do internal mammary artery implants in July, found that 80 per cent of his arteries were blocked in the first two months of his experience. After he had learned to implant the artery according to a technique which has been laboriously worked out over a number of years in this center, 86 per cent of his arteries remained open.

For those who find internal mammary artery blocks in a high percentage of experimental animals, I would suggest that they visit our laboratory, where our experimental Fellow will be pleased to show them the various technical points we have learned. Dr. William Bigelow did this two years ago, and has conveyed to me the fact that practically all his implanted mammary arteries in animals remained open. Dr. Bigelow had added one other feature to the technique—namely, that of wrapping the internal mammary artery in the medial pericardial fat pad. This appears to prevent the artery from angulating and from adhering by scar to the neighboring structures.

The injection technique used by Dr. Neptune is most difficult to evaluate as the injection mass was introduced into the internal mammary artery while the heart was beating. It is well known that in order to demonstrate anastomotic channels in the ventricles of either human or animal hearts, it is necessary to place the hearts on ice for at least twenty-four hours in order to get rid of the spastic elements that occur immediately after death. Many others have confirmed the frequency of internal mammary artery anastomosis, and have found that the facts as given by us are approximately the true situation.

We have found that the frequency with which the internal mammary artery joins up with the arterioles in the left ventricle is dependent upon the condition of the heart at the time the artery is implanted. For example, when placed in normal hearts one cannot expect more than approximately 46 per cent anastomoses. When placed in an ischemic heart, the anastomosis rate is increased to around 76 per cent. The internal mammary artery will stay open, without necessarily giving out large branches, in more than 86 per cent of all implants when the proper technique is followed.

In those animals in which coronary artery insufficiency had been produced by wrapping Cellophane around the anterior descending branch of the left coronary, the implanted internal mammary arteries remained open in every instance.

There is no question that there will be some intimal proliferation; however, this usually occurs distal to the open sixth intercostal artery. The portion distal to the sixth intercostal is not in use, and therefore tends to block off by intimal proliferation just as the ductus arteriosus or other vessels in the human body do when their presence is no longer required.

ANATOMIC, PHYSIOLOGIC, AND SURGICAL CONSIDERATIONS IN REPAIR OF INTERATRIAL COMMUNICATIONS IN MAN

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OPERATIVE repair of atrial septal defects is now feasible, largely because of the efforts of Cohn,[1] Swan,[2] Murray,[3] Bailey and associates,[4] Lewis and Taufic,[5] Gross and co-workers,[6] and others. It is important, however, to clarify the indications for operation, to select the best methods of repair, and objectively to assess the results. The necessity for exact diagnosis has resulted in a clearer concept of the hemodynamic aspect of the condition, but certain problems require further elucidation. Our experience to date with the anatomic, physiologic, and surgical aspects of interatrial communications is presented.

ANATOMIC VARIANTS IN ATRIAL SEPTAL DEFECT

Problems of surgical repair demand an exact knowledge of the anatomic variants of interatrial communications. A classification of these communications is presented in Table I.

TABLE I. CLASSIFICATION OF INTERATRIAL COMMUNICATIONS

I. Normal interatrial communication: valvular-competent patent foramen ovale
II. Abnormal interatrial communications
A. Valvular-incompetent patent foramen ovale
1. With complete posterior septal rim
2. With absent posterior rim and "secondary" anomalous pulmonary venous connection of right lung
3. With associated congenital anomalous pulmonary venous connections
B. Defects of the lower portion of the atrial septum as part of persistent common atrioventricular canal
C. Communications of unusual location or nature

Valvular-Competent Patent Foramen Ovale.—An interatrial communication is present in about 25 per cent of normal people, the so-called valvular-competent patent foramen ovale. This has been termed the "probe-patent" foramen ovale, implying small size, but in adults the tract may measure 1 cm. or more in diameter. In normal persons, the pressure in the left atrium exceeds that in the right atrium; hence the valvular flap is kept closed. Under these

From the Mayo Clinic and Mayo Foundation, Rochester, Minn. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

Read at the Thirty-fourth Annual Meeting of the American Association for Thoracic Surgery, Montreal, Quebec, May 3 to 5, 1954.

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circumstances, left-to-right shunts cannot occur. In congenital or acquired conditions wherein the right atrial pressure exceeds that in the left atrium, right-to-left shunts develop across these normal interatrial communications.

Valvular-Incompetent Patent Foramen Ovale.—A valvular-incompetent patent foramen ovale is present in some persons. This is an abnormal inter-

Fig. 1.—Normal heart viewed from front, showing proximity of orifices of right pulmonary veins (black probes) to the atrial septum. Orifices of left pulmonary veins are far removed from the septum. This anatomic arrangement permits preferential shunting of blood from right lung across atrial septal defects. Orifice of inferior vena cava (I.V.C.) lies close to fossa ovalis. C.S. = Coronary sinus.

atrial communication that usually is crescentic in shape and related to an inadequate length of the flap of the original septum primum. However, such defects do vary in size and exact location. Occasionally, instead of being a clean hole, such a defect may have lacy strands of tissue traversing it. The orifices of the right pulmonary veins in the posterior wall of the left atrium are near the atrial septum and thus are in close proximity to an atrial septal defect (Fig. 1). The orifices of the left pulmonary veins are considerably removed from the septum, since they connect to the left lateral wall of the atrium.

Many atrial septal defects of this type, although large, possess a complete rim of septal remnant. Some, located near the inferior or superior vena cava, are not bounded by septal remnant at the orifice in question. A special problem is posed by large atrial septal defects of this type in which no rim of septal remnant exists posteriorly. The orifices of the right pulmonary veins, which normally lie near the septum in the posterior wall of the left atrium, in these cases lie to the right of the plane of the septum.[4] This situation may be described as an anomalous connection of the veins of the right lung to the right atrium. The anatomic malformation could be termed atrial septal defect, and "secondary" anomalous pulmonary venous connection.

A true congenital anomalous pulmonary venous connection of the right or the left lung may be associated with this type of atrial septal defect. In contrast to the afore-mentioned "secondary" anomalous connection, congenital anomalous pulmonary venous connections are found at sites predictable on an embryologic basis. An important variant of this type is congenital anomalous connection of the right pulmonary veins to the superior vena cava and cephalic portion of the right atrium.

Defects of the Lower Portion of the Atrial Septum.—Interatrial communications may lie just above the rings of the mitral and tricuspid valves. In such cases, the rings and the valvular leaflets themselves may be cleft and the defect may extend down to involve the upper portion of the ventricular septum; this condition is called "persistent common atrioventricular canal."

Communications of Unusual Location or Nature.—Rare communications, such as by way of a coronary sinus with a connection to the left atrium, will not be considered.

PHYSIOLOGIC STUDIES

Fortunately, these anatomic variations cause specific physiologic findings that frequently permit their preoperative identification. Study by cardiac catheterization in all patients clinically diagnosed as having atrial septal defect and being considered for operation is necessary for precise diagnosis and for localization of the defect.

Determination of intracardiac pressure and oxygen saturation and calculations of systemic and pulmonary blood flow in this study were made by methods previously described from this laboratory.[7-9] Dilution curves of T-1824 were recorded in the arterial system by means of cuvette and ear oximeters after injection of the dye into chambers of the heart and great vessels during cardiac catheterization.[10] The contours of such dilution curves are largely determined by the characteristics of flow through the heart and lungs and are distorted in a specific manner by flow through abnormal vascular communications.[11, 12] In general, dilution curves recorded from patients who have defects characterized by left-to-right shunts have lower peaks of amplitude than do curves recorded from normal persons, while the declining concentration of dye after the peak is disproportionately prolonged due to repetitive recirculation of a portion of the dye through the heart and lungs with each circulation.

Physiologic data from 53 patients who had atrial septal defects and who were studied in the cardiac catheterization laboratory at the Mayo Clinic have been summarized by Weidman and associates[13] (Fig. 2); these data are in essential agreement with the findings of most other workers. They indicate that systemic flow is typically normal in this condition in the absence of cardiac failure, but that pulmonary blood flow is characteristically increased. In most instances, the pulmonary arterial pressure is a little above the normal range.

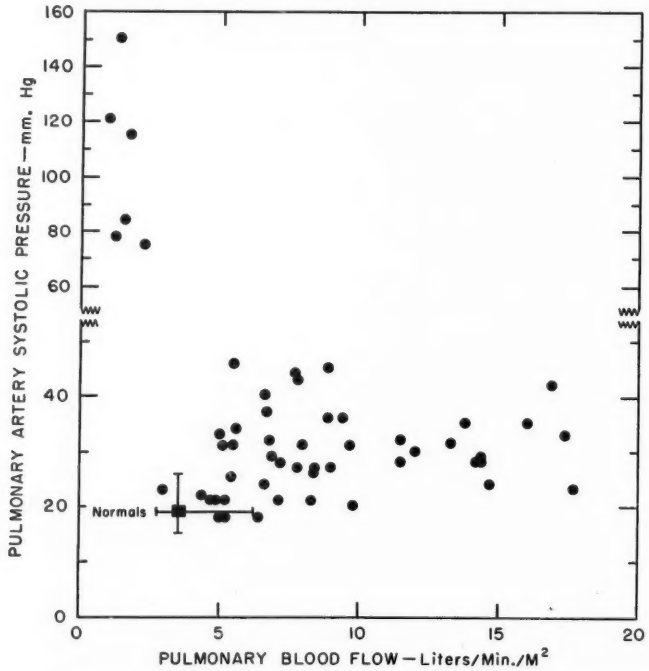

Fig. 2.—Relation of pulmonary arterial systolic pressure to pulmonary arterial blood flow in 53 patients who had atrial septal defects.[13] The average value for 23 normal persons is represented by the solid square; the lines extending from this square show the ranges of normal. A moderate increase in pulmonary systolic pressure that may not be correlated with increasing flow is usual in patients who have atrial septal defects. Note that pulmonary flow is actually reduced in patients who have pronounced increase of pulmonary arterial pressure, an exception being Case 11, studied after these data were compiled.

In uncomplicated atrial septal defect, the flow across the defect is predominantly from left to right and is often of great magnitude. By means of the indicator-dilution technique it has been possible to demonstrate preferential flow of blood through the defect from the right lung in cases of defects of the type characterized by a valvular-incompetent patent foramen ovale without either "secondary" or congenital anomalous pulmonary venous connection.[14] Thus, in the usual case of atrial septal defect, most of the blood streaming into the left atrium from the left pulmonary veins goes through the

adjacent mitral valve into the left ventricle, while a smaller amount is shunted across the septal defect. In contrast, a greater proportion of the blood entering the left atrium from the right pulmonary veins flows across the anatomically adjacent defect in the atrial septum, and a smaller amount continues

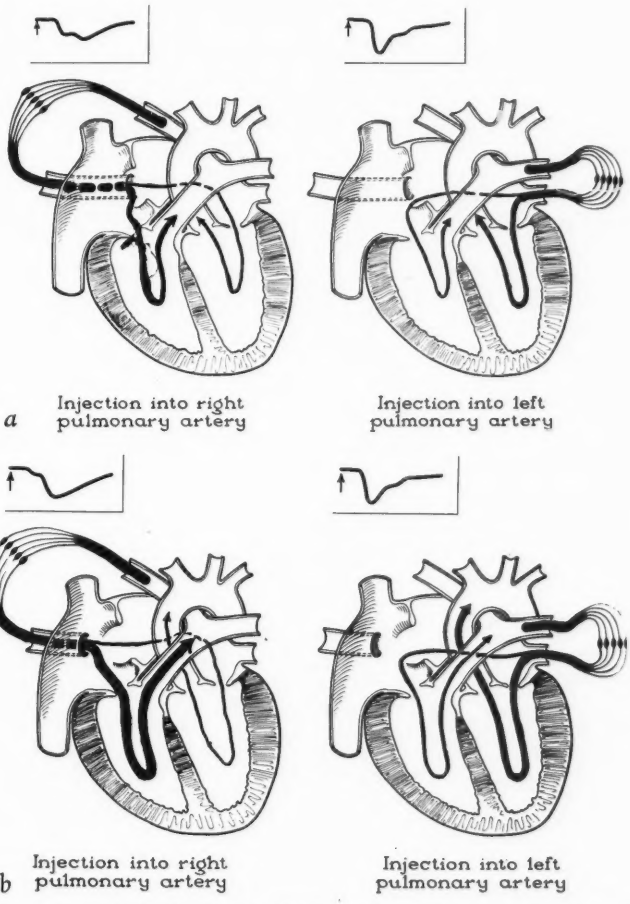

Fig. 3.—Left-to-right shunts through atrial septal defects; diagrammatic representation of path taken by dye injected into the right and left pulmonary arteries. A solid circle indicates the site of injection; the typical contour of the resultant dye-dilution curve is inscribed above each diagram. The instant of injection of dye is indicated by a vertical arrow. *a*, When dye is injected into the right pulmonary artery, the greater proportion of it usually passes into the right atrium through the atrial septal defect, presumably because of the anatomic relations described in the text and illustrated in Fig. 1. When dye is injected into the left pulmonary artery, the greater proportion passes through the orifice of the mitral valve and out into the systemic circulation. The dye-dilution curves differ in that when dye is injected into the former site, the initial deflection of the curve is small because of the reduced contribution of blood from the right lung to the systemic circulation, the greater proportion of it passing across the atrial septal defect to recirculate through the lungs; when dye is injected into the left pulmonary artery, the converse is true. *b*, Circulatory path in atrial septal defect with "secondary" anomalous pulmonary venous connection. Almost all of the dye injected into the right pulmonary artery passes through the right atrium to circulate again through the lungs; an extremely small proportion of blood passes through the left atrium and thence to the systemic circulation.

across the left atrium to pass through the mitral valve (Fig. 3, *a*). The disposition of the veins in the posterior wall of the left atrium, previously mentioned, offers ready explanation for this finding.

In cases of atrial septal defect with absence of the posterior rim associated with "secondary" anomalous pulmonary venous connection of the right lung, indicator-dilution curves recorded after injection of dye into the right pulmonary artery indicate that an extremely small amount of blood from the right lung goes to the left atrium and that almost all of it traverses the right atrium to pass through the tricuspid valve and recirculate through the lungs (Fig. 3, *b*). In instances of associated congenital anomalous pulmonary venous connection, the dilution curves recorded after injection of dye into right or left pulmonary arteries indicate that practically all the blood from the anomalously connected lung passes into the right atrium and thence again to the pulmonary circulation (Fig. 4, *a*).

Although the over-all shunt in most cases of atrial septal defect is from left to right, there is frequently a small right-to-left shunt of blood entering the heart from the inferior vena cava,[15] which lies close to the defect. This has been demonstrated by injection of T-1824 into the inferior vena cava. In certain cases, a small right-to-left shunt also occurs from the superior vena cava (Fig. 4, *b*) which lies relatively far from the defect.

A separate category consists of cases in which there is predominantly right-to-left flow across an interatrial communication with consequent cyanosis. This group usually includes atrial septal defect with severe pulmonary hypertension, atrial septal defect with pulmonary stenosis, and atrial septal defect with Ebstein's malformation of the tricuspid valve.

SELECTION OF PATIENTS FOR OPERATION

Certain broad indications for operation in patients who have atrial septal defect are recognized in spite of the small numbers of patients who have been studied and operated on. Conclusions in this regard must be considered tentative and indubitably will require revision in the light of further experience.

Patients who have a left-to-right shunt in excess of 50 per cent of the pulmonary blood flow are considered as possible candidates for operation. Surgical intervention appears particularly indicated when symptoms are present or when the shunt is even more excessive.

When the pulmonary arterial pressure is considerably increased, with systolic values greater than 80 mm. Hg, large left-to-right shunts are usually absent; indeed, in such instances, either a mixed or a completely right-to-left shunt may be present. In a case recently studied (Case 11 of this report) a large left-to-right shunt occurred in the presence of severe pulmonary hypertension. Our experience in this case and the findings of Gross and Watkins[16] suggest rather strongly that attempted closure of an atrial septal defect by the atrial-well technique in the presence of pronounced pulmonary hypertension is accompanied by great risk to the patient.

As already indicated, atrial septal defects occur in association with pulmonary stenosis in some patients. Usually the flow across these defects is

from right to left, but several cases of bidirectional or pure left-to-right shunts have been demonstrated.[17] In the latter situation, consideration should be given to closure of the defect at the time of pulmonary valvotomy if the shunt is large.

Few data are available on which to decide as to the advisability of closure of interatrial communications in which the shunt is from right to left, with consequent cyanosis. In certain carefully selected cases, it may be beneficial to close such defects.

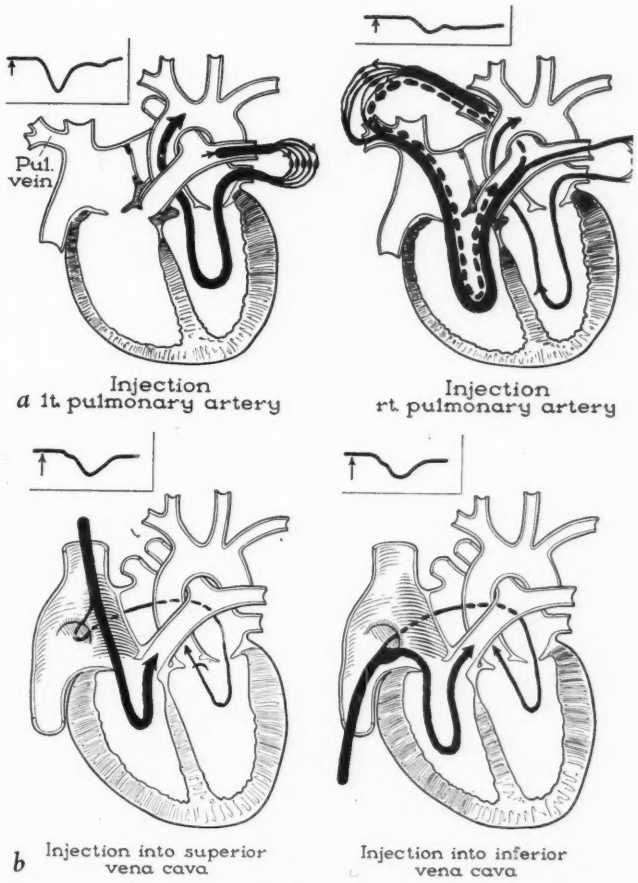

Fig. 4.—*a*, Diagrammatic representation of path of dye injected into the right and left pulmonary arteries in anomalous venous connection of the right lung without atrial septal defect. (See legend of Fig. 3 for details.) Note prolonged appearance time, reduced deflection, and slowed clearance of dye when injection is made into the right pulmonary artery in contrast to the normal curve obtained when dye is injected into the left pulmonary artery.[13] *b*, Circulatory path of dye injected into the superior and inferior venae cavae in atrial septal defect. A small portion of dye crosses the defect into the left atrium and systemic arterial circulation, causing early appearance of dye at the recording site and a "double hump" on the build-up slope of the dilution curve. Note in Fig. 1 the relation of the inferior vena cava to the fossa ovalis, which is a common location for an interatrial communication. This may explain the finding that more inferior caval than superior caval blood usually is shunted.

OPERATIVE TECHNIQUES

Approach.—After the introduction of the atrial well by Gross and associates,[6] we abandoned an earlier experimental technique devised by one of us (Kirklin) and Donald and Grindlay.[18] The well was used extensively in the laboratory and then in the surgical treatment of twelve patients who had atrial septal defects. This technique has proved to be a satisfactory method of approach for repair of these lesions. Since we have not utilized the other methods of approach that have been employed in humans,[2, 4, 5, 19] we are not in a position to compare them with the technique employing the atrial well.

The thorax is opened through the right fifth interspace, after a long lateral incision is made. The atrial well is sutured in place with continuous silk sutures, after an interrupted suture is placed at either angle. Self-retaining retractors have not been placed inside the well. Two cubic centimeters of a 0.04 per cent solution of heparin is dripped into the well each minute. The clotting time of venous blood is more than one hour in most cases immediately after removal of the well, so 25 to 50 mg. of protamine is given slowly into a vein at that time. The well was tolerated by the heart without difficulty (except in one case to which reference is made later); accurate work may be done in the right atrium through it.

Method of Closure.—In view of the results of an investigation of this problem in our laboratory[18, 20] and the work of Bahnson and Baker,[21] we have used thin pieces of polyvinyl sponge (ivalon) for closure of these defects. The sponge is sufficiently pliable to follow the contours of the septal defect after being properly sutured into place. Connective tissue from the septum invades the sponge to hold it firmly in place and the surfaces are rapidly endothelialized.[20]

It is important that a large number of sutures, placed close together, be employed to attach the sponge to the remnant of the atrial septum (Fig. 5, *a* and *b*). In regions where the septal rim is absent, such as occasionally occurs posteriorly or near the caval orifices, the sutures must be brought through the heart to the outside, as emphasized by Gross and co-workers,[6] to be tied over small squares of sponge. Otherwise, there may not be a firm attachment of the sponge in such regions.

Special problems are encountered occasionally in placement of the sponge plug. Accurate preoperative studies are of great value in alerting the surgeon to these prior to operation. A persistent common atrioventricular canal was repaired in one case by anchoring a sponge plug in the defect by a number of sutures in the rim of atrial septum; two sutures were placed along the top of the ventricular septum and one was put in the base of the septal leaflet of the tricuspid valve (Fig. 6).

In cases of large atrial septal defects with absence of the posterior rim and with secondary anomalous pulmonary venous connection of the right lung in which this technique is used, the polyvinyl sponge must be sutured into place so that its posterior aspect lies to the right of the venous orifices.[6] Anteriorly, the sponge is made to curve to the left in such a manner that the caval orifices enter the atrium to the right of the sponge. The sutures must be tied outside the heart in the absence of septal remnants, as mentioned pre-

iously (Fig. 7, *a* and *b*). In one instance (Case 10) of atrial septal defect with congenital anomalous pulmonary venous connection of the entire right lung into the lower superior vena cava and high right atrium, a repair was made by utilization of this same principle.

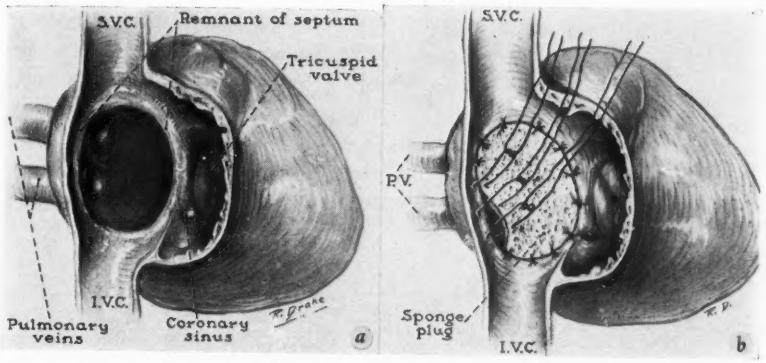

Fig. 5.—Large atrial septal defect (valvular-incompetent patent foramen ovale). *a*, Note complete rim of septal remnant. *b*, Closure of defect by plug of polyvinyl sponge sutured into place by interrupted silk sutures.

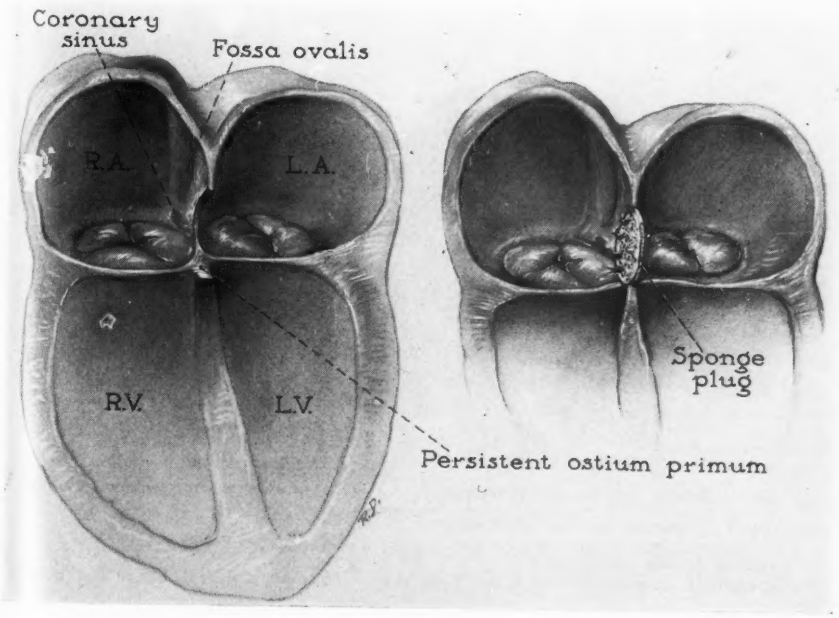

Fig. 6.—Atrial septal defect (partial persistent atrioventricular canal). The defect extends down so that its lower margin is the ventricular septum. The defect was repaired by a sponge plug.

RESULTS

A single death occurred in our previously mentioned group of twelve patients. The remaining eleven patients were alive and in good clinical condition when this article was written. The results are summarized in Table II.

TABLE II. DATA ON 12 PATIENTS SURGICALLY TREATED FOR ATRIAL SEPTAL DEFECTS

DEFECT	CASE (NO. IN SERIES)	AGE (YR.) AND SEX	PREOP. SHUNT (PER CENT*)	POSTOPERATIVE STUDIES†		RESULTS
				INTERVAL POSTOP.	FINDINGS	
Valvular-competent patent foramen ovale with Ebstein's malformation of tricuspid valve	2	30 M	R-L 25	3 wk.	Residual 3 per cent R-L shunt	Excellent
Valvular-incompetent patent foramen ovale without anomalous pulmonary venous connection	3	27 F	L-R 78	7 mo.	Residual 33 per cent L-R shunt also shown in dye-dilution curve	Improved
	8	30 F	L-R 70	2 mo.	Very small residual L-R shunt; dye-dilution curve compatible with same	Excellent
	9	39 F	L-R 71	Not yet studied		
	12	29 F	L-R 75	Not yet studied		
	11‡	48 F	L-R 80			Operative death
	5§	23 F	L-R 88	Not yet studied		
	7‖	20 F	R-L 16; L-R moderate	Not yet studied		
Valvular-incompetent patent foramen ovale with "secondary" anomalous pulmonary venous connection	1	43 F	L-R 80	14 mo.	Residual 60 per cent L-R shunt (also shown in dye-dilution curve)	Unsatisfactory
	4	22 M	L-R 80	5 mo.	No residual shunt; dye-dilution curve normal	Excellent
Atrial septal defect with congenital anomalous pulmonary venous connection of right lung to superior vena cava and high right atrium	10	23 M	L-R 67	6 mo.	No residual shunt; dye-dilution curve normal	Excellent
Persistent common atrioventricular canal	6	28 F	L-R 68	6 mo.	No residual shunt; dye-dilution curve normal	Excellent

*R-L shunts = per cent of systemic flow; L-R shunts = per cent of pulmonary flow.

†Cases 2 and 10 studied by dye-dilution curves only; Cases 1, 3, 4 and 8 studied by dye-dilution curves and cardiac catheterization.

‡Pulmonary hypertension also present.

§Mitral stenosis also present.

‖Congenital pulmonic stenosis and L-R shunt across atrial septal defect also present.

The death (Case 11) occurred in a patient who had a large atrial septal defect and severe pulmonary hypertension, with a pulmonary arterial systolic pressure of 120 mm. Hg and an elevated right atrial pressure. Although it was

difficult to make the decision to operate, the fact that there was persistence of a large left-to-right shunt and that the patient's clinical status was serious made an attempt at surgical correction advisable. After application of the well and while the sutures were being inserted into the septal remnant, cardiac failure ensued and the patient died.

In Case 2, a valvular-competent patent foramen ovale and Ebstein's malformation of the tricuspid valve allowed a right-to-left shunt at the atrial level, with consequent cyanosis. The foramen ovale was closed by approximating the valvular flap with a single suture. Because of the right-to-left shunt in this patient before operation, the oxygen saturation of the systemic arterial blood was 86 per cent at rest; it decreased to 60 per cent on exercise.

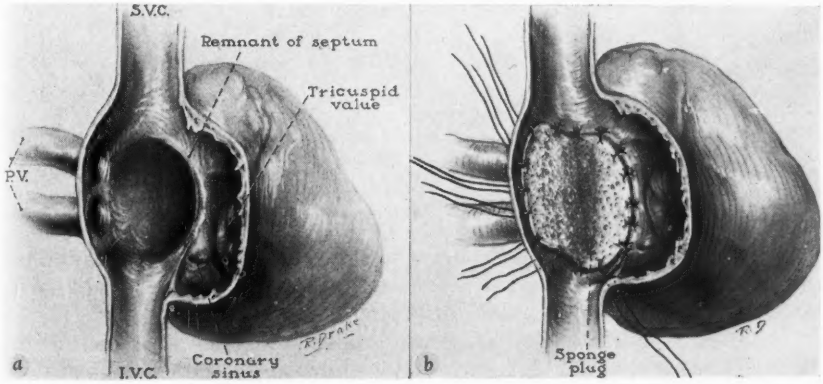

Fig. 7.—Large atrial septal defect (valvular-incompetent patent foramen ovale) with absent posterior rim and "secondary" anomalous pulmonary venous connection of right lung. *a*, Note that the orifices of these veins are in a plane to the right of the orifices of the venae cavae. *b*, Repair by a sponge plug, so fashioned that it diverts the caval flow into the right atrium and flow from the right pulmonary vein into the left atrium.

After operation, the systemic arterial oxygen saturation was 97 per cent at rest and 92 per cent on exercise. A dye-dilution curve indicated that a small (3 per cent) right-to-left shunt still persisted after operation. Symptomatic improvement has been pronounced.

In Cases 3, 5, 7, 8, 9, and 12, large (approximately 4 by 6 cm.) atrial septal defects classed as valvular-incompetent patent foramen ovale without anomalous venous connection were closed with plugs of polyvinyl sponge. In Case 3, a good closure was considered to have been obtained at operation. At cardiac catheterization seven months later, a significant left-to-right shunt was still present; however, the magnitude of this shunt had been reduced from 78 to 33 per cent of pulmonary flow (Table III). Obviously, the sutures in this case must have given way at several points to allow separation of the sponge from a portion of the septal rim. One patient (Case 8) was studied two months after operation by the dye-dilution technique with injection into the superior vena cava and by cardiac catheterization. Closure was nearly complete, but a very small left-to-right shunt persisted. As the result of these

observations, even greater care has been taken in the other cases to anchor a large number of sutures securely in the septal remnant. Thus, in the remaining four cases of this group, secure closure was thought to have been obtained, but postoperative studies have not been made as yet. In one of these four cases, mitral valvotomy was done through the defect because of associated mitral stenosis. In another, pulmonic valvotomy was done during the same operation because of congenital valvular pulmonic stenosis with intact ventricular septum.

TABLE III. CARDIAC CATHETERIZATION DATA BEFORE AND AFTER SURGICAL REPAIR OF ATRIAL SEPTAL DEFECT

		CASE 3		CASE 4	
	LOCATION	PREOPERATIVE	POSTOPERATIVE*	PREOPERATIVE	POSTOPERATIVE†
Pressure (mm. Hg)	Right atrium	15/6	12/5	7/0	8/1
	Pulmonary artery	32/18	26/16	28/14	26/14
Blood flow‡	Systemic	3.0	4.2	3.1	3.6
	Pulmonary	13.8	6.3	14.2	3.6
Left-to-right shunt, per cent of pulmonary flow		78	33	79	0

*Seven months after operation.
†Five months after operation.
‡Liters per minute per square meter of body surface.

In Case 6, uneventful convalescence occurred after what was interpreted as complete closure of a persistent common atrioventricular canal and of its associated septal defects. Postoperative studies were normal.

In Case 1, it was known at operation that incomplete correction of the defect had been obtained; this was confirmed by cardiac catheterization fourteen months after operation. In Case 4, a similar large atrial defect with absence of the posterior rim, associated with a "secondary" anomalous pulmonary venous connection, was considered to have been completely repaired at operation. This was verified by cardiac catheterization five months later (Table III).

In Case 10, the contour of the postoperative dye-dilution curve was entirely normal, and no shunts were found 6 months postoperatively.

SUMMARY AND CONCLUSIONS

Surgical repair of abnormal interatrial communications demands exact knowledge of the anatomic variants of such anomalies. Precise study by cardiac catheterization and selective dye-injection techniques often allows accurate diagnosis and localization of these cardiac defects.

A study is presented of twelve patients who had surgical treatment of interatrial communications. Other cardiac anomalies were associated in some instances. The operative technique involved use of an atrial well and plugging of the defect with polyvinyl sponge. Only one death occurred in this series, and that took place during operation on a patient who had severe pulmonary hypertension.

Tentative criteria are given for the selection of patients for surgical correction of interatrial septal defects.

ADDENDUM

We have now operated on 24 patients with atrial septal defect with predominantly left-to-right shunts. The only death in this group is the one already reported in this paper.

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DISCUSSION

DR. WILLIAM L. JAMISON, Philadelphia, Pa.—I should like to give you our experience with 47 cases of atrial septal defect that we have closed at the Bailey Clinic.

(Slide) To palpate the defect in one case an ungloved finger is inserted through the auricular appendage. An anchoring suture is then placed to approximate the auricle to

the remnants of the septum. Next, a line of continuous suture is inserted following the first suture that was placed to anchor the auricle to the septum. Here the defect is completely closed. The auricle has been sutured to the remnants of the septum and the opening through which the finger is inserted is now closed. This creates a patch which closes the defect. This is the first case treated in this manner. The shunt was entirely closed; preoperatively it was 13.2 liters, postoperatively it was zero.

In cases where there are anomalous pulmonary veins the defect may be closed in a similar fashion. This particular idea was first suggested by Dr. Neptune. At that time he was interested in closing an atrial septal defect, but I expect now he will be spending some time with Dr. Vineberg.

In cases where the defect is very large, a passageway is created between the superior and the inferior vena cava to allow blood to get to the tricuspid valve.

The mortality in cases of ostium secundum has been 9 per cent. In ostium primum the mortality has been much higher, but we are lowering this mortality considerably in our continuing work.

We have found this technique applicable in all cases and very satisfactory.

DR. ROBERT E. GROSS, Boston, Mass.—I think that Dr. Kirklin and his associates have done a fine job in the study and care of their patients.

To date we have operated upon 30 cases of interatrial septal defects, with direct suture closure or by the onlay of a polyethylene sheet. Our approach at present is to explore the right auricle with the left index finger; if there is a posterior defect which is not too high or too low, one can place a straight row of stitches, which brings in the back wall of the auricle to the anterior edge of the septum, and get a closure effectively and quickly. If we happen to find an orifice which is not very large, and not too high or too low, we use the Bailey technique. We have had some good results with it. If we find a hole which is very high against the orifice of the superior vena cava, or low down near the tricuspid valve, we prefer to lay on a polyethylene sheet to cover the defect. To date, one-half of our cases have been treated by the "well" approach, and the remaining one-half by the other forms of closure just indicated.

We have abandoned the use of Pronestyl both before and during operation. We gave up Novocain in the pericardium long ago, because of the depressing effects on the general cardiac mechanism. There is, however, a place for Novocain. In some cases the auricle is quite irritable, and working on it may produce many extra systoles or a run of tachycardia; we have found that by swabbing 2 per cent Novocain on that part of the auricular wall on which we will be working (without allowing the fluid to run down onto the remainder of the heart and thereby depressing it), one can get rid of most of these disturbing irregularities. We have very little confidence in digitalis, but we insist upon quinidine. We feel this drug helps many of these hearts to get through this surgery with a minimum of disturbance. If, during operation, we find that a heart is flagging or weakening, we use rather liberally 5 or 6 c.c. of 10 per cent calcium chloride directly into the heart, and 20 to 30 c.c. of 50 per cent glucose. We have no proof that these help, but we have a strong feeling that they have prevented many catastrophes.

While some of the older patients have recovered promptly from this type of surgery, four or five patients in the late thirties or forties, in spite of the fact that they have had an excellent closure of the defect, have required long periods of rehabilitation to get back into acceptable cardiac status. These hearts are "tired," and it takes a good deal of treatment to get them into satisfactory condition again. In contrast, children or young adults who have closure of septal defects exhibit a prompt and satisfactory recovery. These points lead us to believe that all young patients who have large shunts (despite the fact that they may have no symptoms at the time) should be operated upon. If the right ventricular output is twice or more than that of the left ventricle, we think operation should be undertaken.

DR. JAMES L. SOUTHWORTH, Knoxville, Tenn.—Dr. Kirklin has very properly emphasized the hemodynamic side of the problem, where more work will need to be done.

On the other hand, there has been much technical searching, I think, for a method of operation to close atrial defects. After hearing Professor Crafoord last fall, my associate, Dr. Dabbs, and I became interested in trying his method to close atrial defects, and we tried it in animals. The essential feature, as you know, is to dissect a groove between the two atria and sew this to the intraventricular septum.

(Film) This shows the beginning of a dissection between the right and left atria. The pulmonary vein is at the bottom of the picture and the tissue is held there by a forceps. It was surprising to me how easy it is in animals to dissect the two atria. It is possible to clear the superior vena cava very far behind and to see the aorta there. The finger is now within the atrium, and the suture will be passed into the intraventricular septum under finger guidance. There the needle came out a little too high, was withdrawn, and came out in the proper place where the groove was dissected. The animal was sacrificed and left in the same position; the needle was passed again into the intraventricular septum.

None of the eight dogs we operated on had difficulties during the operation; all survived except the one we sacrificed. Thus, we were encouraged to try this operation on a 32-year-old woman who had a very large defect of the septum primum type, about 8 cm. in diameter; we had no difficulty in closing this defect, and the patient has made an uneventful recovery.

DR. RALPH A. DETERLING, New York, N. Y.—I think the work presented here both by the essayist and the discussants is excellent. I want to bring up two points from our experience. We have been faced with operating on desperately ill children. In this group we have encountered either large septum primum defects in which there is just a little ridge between the two valves as shown in Dr. Kirklin's last case, or else an atrioventricularis communis where a good-sized defect is also present between the valves and in the upper portion of the ventricular septum. Although we have had poor results in these cases, they do need surgery. I think it is important to emphasize that some of the patients who most need surgery still pose a great technical problem.

I mention one case because of the difficulties we encountered. There was thought to be a mitral insufficiency in addition to a large atrial septal defect. The pressure in the right auricle was 44 mm. Hg. We could not use a rubber well, since the blood would come up over the top. We invaginated the atrial wall by the Bailey technique, and each suture hole spurted blood vigorously, requiring a total of about 3,000 c.c. of blood during the operation in this 9-year-old girl.

DR. FRANK L. GERBODE, San Francisco, Calif.—I would like to compliment Dr. Kirklin on the excellence of his presentation and on the manner in which he closed these defects so successfully. After experimenting with the well technique in the laboratory for a time, my associates and I thought there must be a simpler way of closing the smaller defects. Therefore, about two years ago, we began exploring the possibility of utilizing the plane between the right pulmonary veins and right atrium as a means of obtaining posterior atrial wall for closure, believing it would be better to close the defect posteriorly than to oppose the anterior wall as suggested by Bailey. We found, as Dr. Crafoord did, that one could pass sutures beneath the superior cava and beside the aorta, and thus close the defect posteriorly. However, there are some disadvantages in this method of placing the sutures. I would like to present another method for suturing which might be called the "hook" technique.

(Slide) Most of the infolding techniques involve pushing down the lateral or anterior atrial wall, or auricular appendage to fill the defect. I have found that if a sharp hook with a slender shaft is passed through the lateral atrial wall, and guided to the margin of the defect with the left index finger placed in the auricular appendage, one can engage the medial margin and pull it in apposition to the lateral margin and lateral atrial wall. An assistant can then hold the hook, with the margins thus held together, while a suture is passed through them from the outside. The first suture should be placed at the lowermost part of the defect, and the closure made from below upward.

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(Slide) This shows the use of the hook as an aid in the closure of a larger defect, where the lateral atrial wall is used as a circular patch. Again, the closure should start at the inferior margin, gradually approximating the edges in a circular manner.

This is a very simple method and should be useful for all defects save the very large ones. When combined with dissection of the posterior atrial plane a minimum of distortion results. It should be remembered, however, that great variations exist in atrial septal defects, and when the pulmonary venous return is abnormally placed, the dissection may not be possible.

DR. WILLIAM H. MULLER, JR., Los Angeles, Calif.—I would like to compliment Dr. Kirklin on the very fine work he has presented here, and I would like to mention briefly our own experiences in dealing with this problem. We have used primarily the procedure developed by Dr. Gross, because it gives one an opportunity to explore the interior of the auricle leisurely. Furthermore, one is not faced with the problem of air emboli. We have been able to approximate the edges of the defect by direct suture in all instances except one, which was in a patient with a septum primum; here we employed a nylon plastic patch, but about three weeks later we had to remove it because she developed severe cardiac failure.

We have operated on 17 patients and have had 2 deaths—one a 4-year-old patient who had been in severe failure and one a 30-year-old patient with severe pulmonary hypertension. We think the most critical point in the operative procedure is when one opens the well. We routinely use an indwelling cannula in the brachial artery to measure continuous direct pressures; often the pressure will drop to almost zero shortly after the clamps are removed from the auricle. Following the rapid introduction of blood through the cannula the pressure usually becomes elevated and the heart action becomes stable.

(Slide) This slide shows some of the physiologic studies which we make on these patients during operation, before and after the defect is closed. There has been a drop in pressure in the right auricle and a rise in pressure in the left auricle, as one would expect. There is also a diminished pressure in the pulmonary artery and a rise in pressure in the aorta. The oxygen saturation does not drop quite as much as one would expect in the right auricle, but these patients are receiving high concentrations of oxygen.

DR. F. JOHN LEWIS, Minneapolis, Minn.—I should like to call attention to another variation in the anatomy of this complicated defect that we have encountered in closing them under direct vision with the aid of hypothermia.

(Slide) I think this variation is about as common as anomalous pulmonary venous drainage. Four of the last seven patients we have operated on have had it. It concerns the position of the valve of the inferior vena cava, which is normally located to the right of the vena cava. In this variation of the defect there is no lower rim, instead the valve of the inferior vena cava forms a false lower rim. If repaired improperly, blood from the inferior vena cava would enter the left atrium after closure.

(Slide) After the first stitch is placed and tied down, the rest of the repair is easily accomplished with a running stitch. In these cases, as well as in those with anomalous pulmonary venous drainage, we have found that simply stitching the edges together has been satisfactory, though in many cases it is very important to place the stitches in the right position to form new margins. Grafts have not been necessary, perhaps because when the heart is empty and collapsed the edges fall together satisfactorily.

Our experience with infants and young children has been the poorest we have had. We have operated on 16 patients whom we thought preoperatively had atrial septal defects. Three infants were found to have anomalies that we considered to be inoperable at the time. Two had complete A-V canals and one had a ventricular septal defect. None of the 3 survived. Of the 13 remaining, one patient did not have his operation completed, although he recovered. Another patient had a septum primum defect and died three days postoperatively of complete heart block. Of the remaining 11 patients one died because of a technical error, leaving 10 successfully treated cases. Among the 12 patients who actually proved to have atrial septal defects, 4 had the type of anomaly just mentioned,

4 had anomalous pulmonary venous drainage, and one had a septum primum defect. There were 2 with complicating mitral stenosis. Actually only 3 had what one would consider to be a simple hole between the two atria.

DR. EARL H. WOOD (Closing): First, I must admit I am a physiologist and not a thoracic or cardiac surgeon, so it is obviously impossible for me to comment on the surgical considerations in this discussion.

I would like to emphasize that atrial septal defect is often a complicated condition. In the last seven years we have studied approximately 130 cases in which communications between the right and left atrium were demonstrated by the presence of abnormal shunts in one or the other direction. These cases were studied by what might be called special techniques: namely, rapid sampling via the catheter with the cuvette oximeter which allows identification, localization, and accurate measurement of the magnitude of the left-to-right shunts, and by selective arterial dilution curves which allow identification, localization, and accurate measurement of right-to-left shunts. Recently, because of our interest in the surgical correction of this condition, Dr. W. H. Weidman and Dr. H. J. C. Swan very carefully analyzed catheterization records in our laboratory from more than 100 cases; their results emphasize that this is not a simple condition. Of these cases only 60 were found in which the atrial septal defect was the only anatomic anomaly. So, certainly, we agree with the other commentators in the conclusion that this is often not a simple defect.

Even when present as a single defect the problems of correction differ from many other cardiac conditions amenable to surgery, such as patent ductus arteriosus and mitral stenosis, where the variation is only one of degree. In atrial septal defect not only is there a variation in the size and type of hole, but also in the position of the defect in the septum and the relation of the septum to the pulmonary veins.

With special techniques, especially arterial dilution curves, I have been surprised and gratified at our success in being able to predict for Dr. Kirklin the approximate size of the hole and its position in the septum and relation to the pulmonary veins. As you may know, most patients with atrial septal defect show a small right-to-left shunt. If this is very predominant from the inferior vena cava and absent from the superior vena cava, it is good evidence that the defect is low in the atrial septum in the fossa ovalis region where inferior vena cava blood impinges when emptying into the right atrium. Studies of the left-to-right shunt, particularly the degree of preferential flow from the right lung, allow prediction of the presence of an anomalous venous connection or of absence of the posterior rim of the septum. In the majority of cases we have been able to predict quite accurately for Dr. Kirklin what he may expect to find at operation.

AORTIC ARCH RESECTION AND GRAFTING FOR ANEURYSM EMPLOYING AN EXTERNAL SHUNT

ALLAN STRANAHAN, M.D., RALPH D. ALLEY, M.D. (BY INVITATION), ALBANY, N. Y., WILLIAM H. SEWELL, LIEUTENANT (MC) USNR (BY INVITATION), BETHESDA, MD., AND HARVEY W. KAUSEL, M.D. (BY INVITATION), ALBANY, N. Y.

THE procedure of choice in the surgical management of aneurysms is excision with restoration of normal blood flow in the parent vessel. The application of this ideal to aneurysms of the thoracic aorta has heretofore been largely restricted to sacciform aneurysms susceptible of local excision and lateral aortorrhaphy.[1-4] The frequent extension of this principle to the management of aneurysms requiring temporary occlusion of the thoracic aorta to permit safe local excision or resection and grafting has awaited the development of an adequate means of diverting the blood flow during the period of aortic obstruction. The purpose of this report is the presentation of two cases of aneurysm of the thoracic aorta treated by surgical excision and grafting, in which the aortic blood flow was diverted through a shunt of large caliber for periods of six hours and forty-five minutes, and three hours and twenty minutes, respectively.

CASE REPORTS

CASE 1.—*Fusiform and Saccular Syphilitic Aneurysm of Entire Thoracic Aorta.*—M. J., a 53-year-old white man, entered the Albany Hospital on Oct. 22, 1953, because of extreme dyspnea. For a number of years the patient had noted a dry cough and exertional dyspnea. During the six-month period prior to admission cough and dyspnea had increased. Hoarseness and moderate dysphagia ensued, and a 20-pound weight loss occurred. An abrupt and marked increase in his symptoms occurred two weeks prior to admission, forcing him to stop work as a railroad handy man. His cough became productive of purulent sputum which was increasingly difficult to raise, and cyanosis developed. When admitted to the Albany Hospital, he had required oxygen therapy continuously for thirty-six hours. The patient denied chest or back pain. No history of primary syphilis could be obtained.

Physical examination revealed an acutely ill, apprehensive, white man in severe respiratory distress, mildly cyanotic despite nasal oxygen, and unable to raise the audible secretions in his tracheobronchial tree because of respiratory obstruction. The temperature was 100° F., pulse 100, respirations 30, and blood pressure 120/70 in each arm. The right lung field was resonant with bronchovesicular breath sounds and a prolongation of the expiratory phase of respiration. The entire left hemithorax was hyperresonant with markedly diminished breath sounds and sibilant rhonchi throughout. The heart was not enlarged, its rhythm was regular, and no murmurs were heard. The remainder of the examination was normal.

Serologic tests for syphilis were positive. The white blood-cell count was 21,800 with 96 per cent neutrophil forms. The electrocardiogram revealed a right ventricular hyper-

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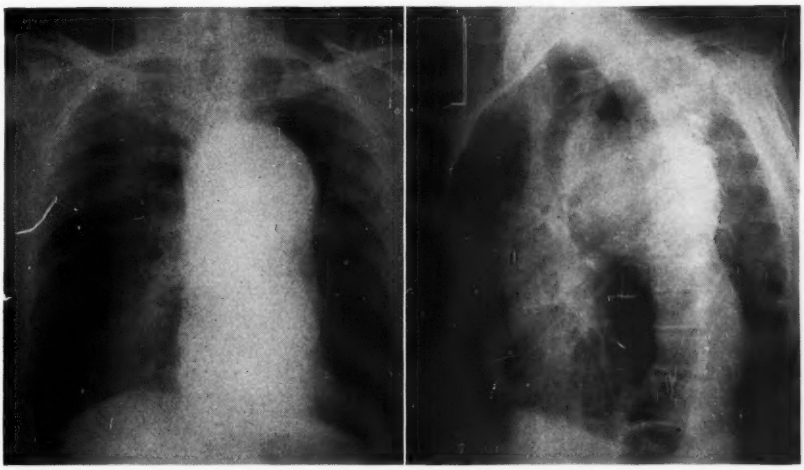
The opinions and conclusions expressed in this paper are those of the authors, and are not to be construed as official or necessarily reflecting those of the Medical Department of the United States Navy, or of the Naval Service at large.

Read at the Thirty-fourth Annual Meeting of The American Association for Thoracic Surgery at Montreal, Quebec, May 3 to 5, 1954.

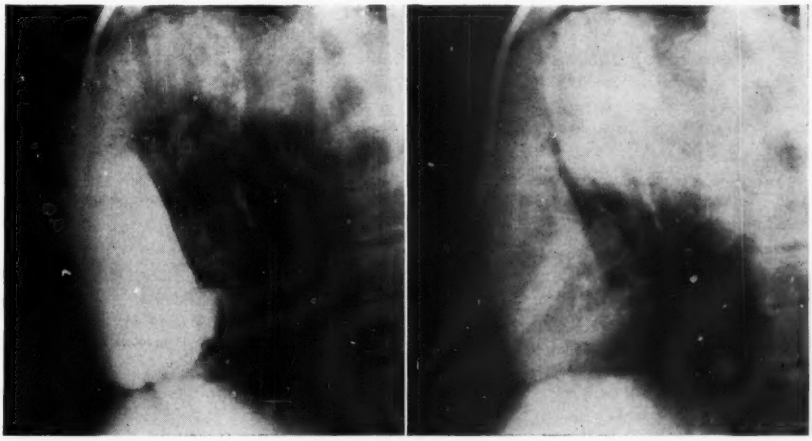
trophy pattern. Other laboratory tests, including urinalysis, bleeding, clotting, and prothrombin times, nonprotein nitrogen, fasting blood sugar, serum proteins and albumin-globulin ratio, and a serum electrolyte pattern were all within the normal range.

Roentgenographic and fluoroscopic examinations (Fig. 1) revealed a large fusiform aneurysm of the aortic arch and descending thoracic aorta with erosion of the third through seventh dorsal vertebral bodies. There was displacement and compression of the distal



A. *B.*

Fig. 1.—Case 1. Syphilitic aneurysm of entire aortic arch in a 53-year-old man. *A*, Roentgenogram of chest showing a large fusiform aneurysm of the arch and descending aorta with obstructive emphysema of the left upper lobe and atelectasis of the left lower lobe. The heart is not enlarged. *B*, Oblique projection revealing anterior displacement of the trachea with compression of the left main bronchus. Vertebral erosion was better seen in the right anterior oblique projection.



A. *B.*

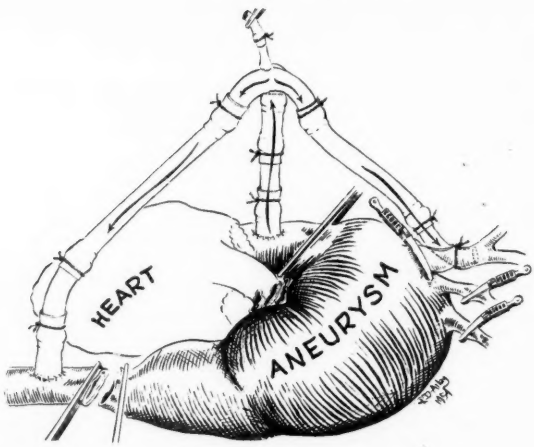
Fig 2.—Case 1. *A*, A retrograde aortogram demonstrates the aortic valve to be competent and delineates the ostia and proximal portions of the coronary arteries which are patent. *B*, A later exposure now shows dye filling the aneurysm and indicates its extent.

trachea and adjacent esophagus. Obstructive emphysema of the left upper lobe and obstructive atelectasis of the left lower lobe were present. A retrograde aortogram delineated the aneurysm more fully. It showed that the aortic valve was competent, and disclosed that the coronary ostia and proximal arterial trunks were widely patent (Fig. 2).

On intensive antibiotic therapy the patient's airway improved perceptibly in the ensuing week, and valuable time was thereby afforded to make preparations for a desperate surgical attempt to retrieve an otherwise hopeless situation.

Operation was performed on Oct. 29, 1953, under general anesthesia, with the endotracheal tube advanced to, but not through, the area of tracheal compression. Thoracotomy was performed through the bed of the left fifth rib with transsection of the sternum and

A.



B.

Fig. 3.—Case 1. *A*, Photograph at operation with left lung retracted showing position and size of aneurysm. At the upper right the innominate and left carotid arteries are seen, the left subclavian is obscured by the phrenic nerve. The pulmonary outflow tract is visible and intimately attached to the aneurysm. *B*, Semidiagrammatic representation of the operation illustrating the shunt in position and functioning. Inside diameter of shunt 10 mm. throughout. Length of shunt limbs: ascending aorta—15 cm., innominate—26 cm., and descending aorta—32 cm. Clamps have been placed proximal and distal to the aneurysm and on the vessels of the arch, with beginning resection of the lesion. *C*, View prior to closure of the chest reveals the aortic graft in position and functioning. The shunt has been removed. All anastomoses are complete. The left lung has been removed.

extension of the incision into the right fourth intercostal space. The aneurysm arose in the ascending aorta and involved the entire arch and descending aorta to the diaphragm (Fig. 3, *A*). The aortic window was completely obstructed by the aneurysm through compression of the left main bronchus, which resulted in obstructive emphysema of the upper lobe and atelectasis with suppurative pneumonia of the left lower lobe. Subsequent dissection disclosed compression of the main pulmonary artery, partial erosion of the compressed distal trachea and the adjacent esophagus, and deep erosion of the third to seventh dorsal vertebrae.

Fig. 3, *C*. *(For legend see opposite page.)*

The proximal aorta, great vessels, and distal descending aorta were freed by dissection to permit attachment of a previously prepared three-limbed shunt to by-pass the area to be resected (Fig. 3, *B*). The shunt consisted of 1 cm. bore Tygon tubing with siliconed glass joints fastened to the ascending and descending aorta by means of heterologous reconstituted freeze-dried arterial sleeves. The latter were anastomosed to the host vessel end to side by employing a Beck-Pott's exclusion clamp. The innominate artery was cannulated with the third limb of the shunt, thereby permitting circulation to the head as well as to the trunk during the period of aortic occlusion. The shunt was then filled with saline solution through the air vent at the top to prevent air embolism and placed in operation. The ascending and descending aorta, left subclavian and common carotid arteries were then occluded, and with the shunt operating, the dissection of the aneurysm itself commenced.

In the course of dissection of the region of the aortic window, an opening was made in the attenuated wall of the left pulmonary artery. This necessitated sacrifice of the left lung, which was already useless because of erosive destruction of the left main bronchus. Except for a proximal and distal cuff, the entire thoracic aorta was then removed. The defect was bridged by two reconstituted heterologous (calf) freeze-dried aortas[5, 6] spliced together. The innominate and left common carotid arteries were anastomosed to the arch thus created end to side. The left subclavian artery was intentionally sacrificed. The new arch was then distended with saline solution to remove all air and the clamps removed from the distal aorta, proximal aorta, and left common carotid artery, in the order mentioned. Distended with blood, the graft was obviously too long and had a tendency to kink. This was corrected by making a sleeve resection of 5 cm. at the level of the previous splice (Fig. 3, *C*) and again employing the shunt during a forty-minute period of occlusion. The reconstructed arch now had an excellent curvature and all suture lines had ceased to ooze. The shunt was therefore removed and the two aortic openings isolated by the exclusion clamp and sutured. The innominate artery was repaired by temporary occlusion

employing two Potts' ductus forceps. During the period of innominate occclusion, circulation to the brain depended upon the left common carotid artery. The chest was then closed with drainage of both pleural cavities connected to a series of water-seal and valve bottles that permitted regulation of the intrapleural pressure at mean minus 10 cm. of water pressure.

The operating time was fifteen hours. A blood loss of 5 liters occurred, but was replaced as lost. The shunt was in operation and the aortic arch excluded for six hours and forty-five minutes. Because the right brachial artery thrombosed after the preoperative aortogram, no record of the patient's blood pressure was available after the left subclavian artery was sacrificed during the operative procedure. The right axillary and temporal pulses, however, remained full with a regular rhythm throughout the operation. The pulse rate ranged between 80 and 120 per minute. Frequent electrocardiographic tracings revealed no signs of heart strain or coronary insufficiency at any time during the operation. At the conclusion of the operation, there were good bilateral carotid and femoral pulses.

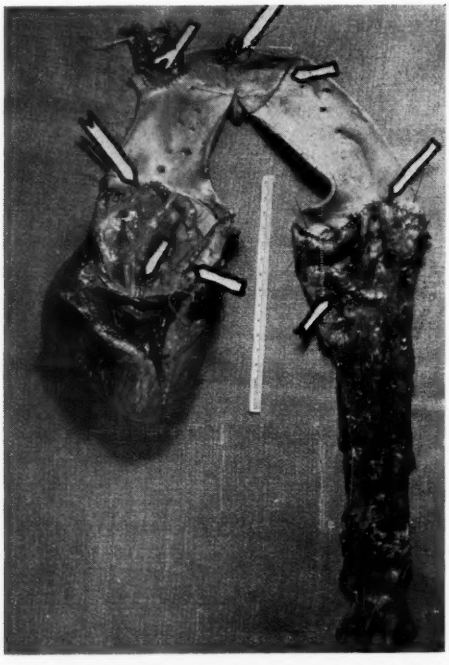


Fig. 4.—Case 1. Autopsy specimen of heart and entire thoracic and abdominal aorta, with longitudinal incision of aorta and left ventricle. Arrow markers from heart peripherally indicate aortic valve, suture closure of proximal shunt limb, proximal anastomosis between ascending aorta and graft, innominate and left carotid anastomoses to graft, graft-to-graft anastomosis, graft and distal thoracic aortic anastomosis, and the closed incision of the distal shunt sleeve aperture.

Following extubation, the patient responded and opened his eyes when addressed. His color was good. He began to thrash around with his left arm and leg, but the right arm and leg were limp. Right intercostal, diaphragmatic, and abdominal muscular function were intact. While the patient was being moved from the operating table, his condition suddenly changed. Immediate reopening of the left chest disclosed that the left pulmonary artery ligature had cut through the vessel and a massive exsanguinating hemorrhage had occurred. The patient did not respond to resuscitative measures. Post-mortem examination revealed all suture lines associated with the graft to be intact (Fig. 4).

Comment.—Aortic arch resection and grafting is a feasible procedure. The shunt employed worked well. The chief objection to the method is the time required to carry out the elaborate procedure. We feel that by improved planning the operating time may be reduced one-third. In the case presented, seven major vascular anastomoses and three vascular suture lines to repair the defects left following removal of the shunt were required.

Except for problems arising from mechanical respiratory obstruction, the patient operated upon was in excellent general physical condition. Significant aortic insufficiency or coronary artery disease were fortunately absent in this patient.

Heterologous freeze-dried grafts were used because human material was not available, and probably would not have been of sufficient size for anastomosis to the dilated proximal and distal aortic cuffs.

Whether the partial right hemiplegia would have been permanent in this patient remains in doubt. It must be assumed to have resulted from ischemia, caused by prolonged interruption of the left carotid and vertebral arteries. Although interruption of these vessels on one side probably is usually well tolerated, the occurrence of partial hemiplegia in Kirby and Johnson's case[14] of innominate aneurysm following cross-clamping of this vessel for fifty-seven minutes, suggests that the maneuver is not uniformly safe. We advocate, therefore, in the future the addition of a fourth limb to the shunt for left common carotid cannulation. This would add but a few minutes to the length of the operation.

CASE 2.—*Classical Traumatic Aneurysm of the Descending Aorta.*[15]—T. W., a 25-year-old Negro man, sustained a comminuted simple fracture of his right femur, simple fractures of the right second and third metacarpal bones, skull fracture, and a mild concussion, together with contusions and abrasions of the chest and abdominal wall, in an automobile accident in September, 1948, five years before admission. The patient was the driver of the automobile and undoubtedly was decelerated by the steering wheel when the vehicle, traveling 85 m.p.h., left the road and struck a tree.

An open reduction of the comminuted fracture of the right femur led to a succession of orthopedic misfortunes which kept the patient in bed or immobilized by plaster spica until September, 1953, when the final cast was removed. A routine photoroentgen film at this time revealed a mediastinal mass projecting to the left.

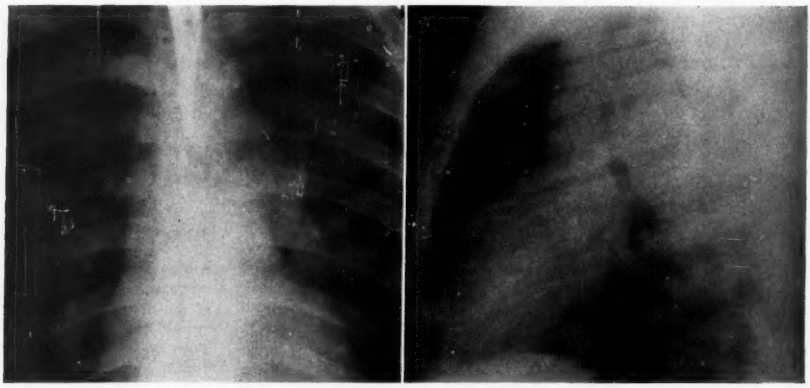
The history disclosed that in high school the patient had been told he had a heart murmur and advised to stop playing basketball. Chest films taken during high school were not explained to the patient. Soon after the automobile accident, the patient experienced short attacks of lancinating pain across his anterior chest. These were followed by the abrupt onset of severe dyspnea which could be relieved by flexing the trunk. No x-ray examinations were made during that period.

Examination following the photoroentgen discovery of the lesion revealed a husky Negro in robust health, but with a distinct limp from shortening of the right leg. The blood pressure was 130/80 in both arms. A Grade 3 systolic precordial murmur with maximal intensity over the pulmonic area, but transmitted to the neck, left parascapular region, and epigastrium, was heard. There was no diastolic component.

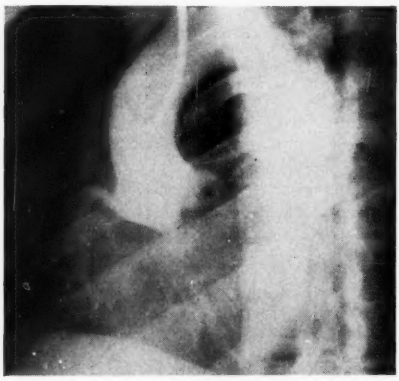
Roentgenographic and fluoroscopic examination revealed a thin, calcified halo encircling an area of nonpulsatile density measuring 7 by 5 cm., inseparable from the first portion of the descending aorta (Fig. 5, *A* and *B*). In the left anterior oblique position, the density appeared to project posteriorly and to deviate the esophagus anteriorly and to the right. A retrograde aortogram proved that the lesion was a thin-walled aneurysm

arising from the first portion of the descending aorta (Fig. 5, *C*). The contrast material remained in the aneurysm for several seconds following complete clearance of the dye from the aorta itself. No patent ductus was shown in the study.

Bronchoscopy revealed indentation of the membranous wall of the left main bronchus and conversion of its lumen into a crescent shape of approximately 70 per cent normal caliber. The electrocardiogram was normal. Serologic tests for syphilis were negative.



A. *B.*



C.

Fig. 5.—Case 2. Posttraumatic aneurysm of the descending aorta in a 25-year-old man. *A*, Roentgenogram of chest reveals a 7 by 5 cm. density in the left hilum rimmed by a thin layer of calcium. *B*, On lateral view the lesion is seen at the origin of the descending aorta. C, A retrograde aortogram with dye filling the aortic arch and the aneurysm. Dye remained in the aneurysm for several seconds after the aorta itself was clear of dye.

Operation was performed Dec. 3, 1953, under general endotracheal anesthesia. Thoracotomy was done through the bed of the resected fourth rib with posterior fracture of the third, fifth, and sixth ribs to obtain adequate exposure. The aneurysm arose just below the left subclavian artery and encircled three-quarters of the circumference of the aorta, to which it was inseparably fused. The dome of the aneurysm was everywhere calcified and very thin-walled. There were no pleural adhesions, but a dense subpleural periaortitis with fusion of the aneurysm to the adjacent pulmonary artery, bronchus, and esophagus rendered the dissection somewhat tedious. A patent ductus of small caliber was ligated and divided.

It became evident that the aneurysm could not be tangentally excised, and that it would be necessary to obstruct the flow in the aorta to permit endo-aneurysmorrhaphy or resection and grafting. Accordingly, a left subclavian-descending aorta shunt, with an inside diameter of 10 mm. abruptly tapered to 6 mm. to fit the subclavian artery, was introduced in the manner depicted in Fig. 6, *A*. With the shunt in operation, the aorta was obstructed above and below the aneurysm and the aneurysm opened. It measured 7 by 5 by 4 cm. and an opening from the aorta measured 2.5 to 3 cm.

The technique of endo-aneurysmorrhaphy as employed by Bahnson[3] was not feasible. Accordingly, the area was resected and the defect bridged by an 8 cm. length of homologous freeze-dried aorta* obtained from a 19-year-old white man, a victim of an automobile accident, shortly after death Aug. 8, 1953. The period of aortic occlusion was three hours and twenty minutes. The effect of the shunt on blood pressure proximal and distal to the area of aortic obstruction was recorded by a Hathaway Electronic Recorder† and is shown transcribed in Fig. 6, *B*. The operating time was eleven hours. Measured blood loss was 3,450 c.c. with a replacement of 4 liters.

There was no postoperative evidence of spinal cord damage. The early postoperative period was marred by the development of oliguria and hypotension related to dehydration and low-salt syndrome which responded promptly to adequate parenteral therapy. Because of recurrent atelectasis of the left lung, on the third postoperative day a temporary tracheotomy was instituted. The patient's subsequent convalescence was uneventful, and he was discharged from the hospital on the twenty-seventh postoperative day. A film of the chest and right brachial and right femoral pulse pressure curves taken five months postoperatively are shown in Fig. 7, *A* and *B*.

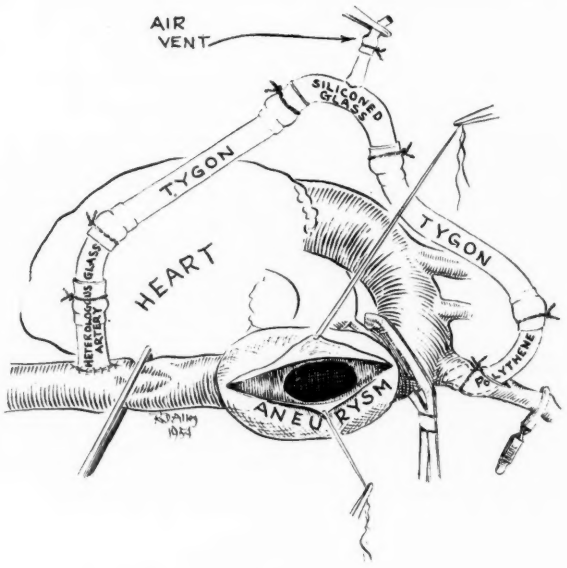
Comment.—Although this patient was free of symptoms referable to his aneurysm, it is known that these lesions carry a poor prognosis.[15] It is conjectured that the long period of immobilization imposed by his orthopedic problems may have played a role in his preoperative survival. We may speculate as to whether the small patent ductus played a significant hemodynamic role in relation to lateral intra-aortic pressures affecting the aneurysm.

The preoperative aortogram gave a true picture of the relatively small size of the opening in the aorta compared to the aneurysm itself. This information, however, was misleading in the final analysis, because the aneurysm was not susceptable of tangental excision or endo-aneurysmorrhaphy. Without the availability of a shunt and suitable graft, an attempted excision of this lesion doubtless would have led to cross-clamping of the aorta and to the attendant risk of permanent neurologic damage.

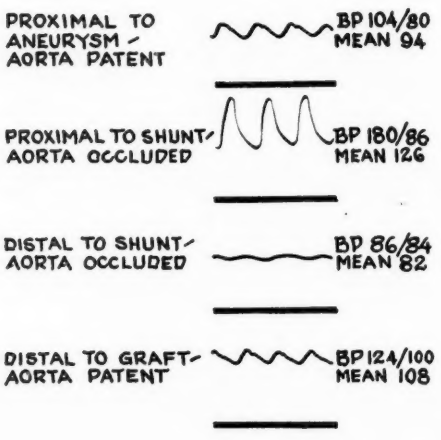
DISCUSSION

Although in the process of resection of aortic aneurysms the descending aorta has been obstructed for periods up to forty-seven minutes without the production of heart failure or of neurologic damage, experimental and clinical evidence indicates that this maneuver is unsafe except in the management of aneurysms complicating coarctation with its attendant expanded collateral circulation.[3, 7-12] The introduction of a shunt to permit diversion of blood flow around an occluded area adds a morbidity factor of its own. In many instances, however, this will be more than compensated for by the time permitted for careful, unhurried dissection and excision of a difficult lesion.

*Supplied by the Tissue Bank, United States Naval Medical School, Bethesda, Md.
†Hathaway Instrument Company, Denver, Colo.



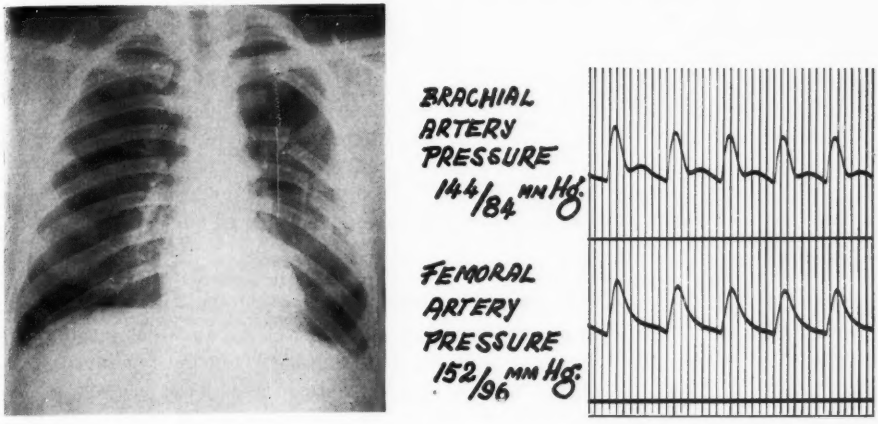
A.


PROXIMAL TO ANEURYSM - AORTA PATENT
BP 104/80 MEAN 94
PROXIMAL TO SHUNT - AORTA OCCLUDED
BP 180/86 MEAN 126
DISTAL TO SHUNT - AORTA OCCLUDED
BP 86/84 MEAN 82
DISTAL TO GRAFT - AORTA PATENT
BP 124/100 MEAN 108

B.

Fig. 6.—Case 2. *A*, Diagram of operation demonstrating position of aneurysm. The sac has been incised and the opening between the aorta and aneurysm depicted. Aorta occluded above and below the lesion with external shunt in place. Also represented is the method of securing the glass joint of the shunt to a sleeve of heterologous freeze-dried vessel (calf) sutured to the aorta. The inside diameter of the shunt was 10 mm. except for the polythene subclavian cannula which tapered to a minimum of 6 mm. at its flanged end. Over-all length of the shunt was 55 cm. *B*, Tracings of records obtained at operation with a 25 gauge needle and recorded by a Hathaway oscillograph. With shunt in operation and the aorta clamped, pressures proximal to the shunt increased. Distally, the pulse pressure was virtually absent, but an adequate mean pressure was maintained. At completion of the operation with the graft in place and the aorta open, normal pressures were registered distal to the graft.

The risk of hindquarter paralysis resulting from interruption of the descending aorta arises from four factors: (1) the level of obstruction (less risk progressing downward from left subclavian artery to abdominal aorta); (2) the duration of obstruction; (3) the number of intercostal artery and other collaterals interrupted in the course of mobilizing the lesion to be excised;



A. *B.*

Fig. 7.—Case 2. *A*, Chest x-ray four and one-half months after operation. *B*, Right femoral and brachial arterial pressures obtained at the same time by intra-arterial puncture and recorded by a Hathaway oscillograph.

and (4) the metabolic rate of the tissues below the level of obstruction. The *combined* effects of high aortic occlusion and interruption of all intercostal arteries were doubtless responsible for the partial paraplegia in Lam and Arm's case[12] following aortic occlusion on two occasions of twenty-four and fifteen minutes, respectively. Conversely, the absence of cord damage following obstruction of the descending aorta for forty-five minutes in DeBakey and Cooley's case[13] may be related to the low level (eighth dorsal) of aortic obstruction in their case.

Hufnagel and Gross,[9] by refrigeration of the backs of dogs, and Beattie and his co-workers[11] employing hypothermia in dogs, prevented hindquarter paralysis following occlusion of the midthoracic aorta for thirty and ninety minutes, respectively. Hypothermia alone, or in combination with shunts of smaller caliber than those necessary at normal rates of metabolism, may be found useful in resectional surgery of the human thoracic aorta. Until measures to minimize the risk of ventricular fibrillation or cardiac standstill in adults under hypothermia are found,[16] however, an external shunt of large caliber would appear preferable for cases requiring prolonged occlusion of the thoracic aorta.

The problem of aortic arch resection employing multiple small-bore (1.14 to 2.69 mm.) polythene shunts has been studied experimentally by Hardin and associates[17] and these authors report a technically achieved but unsuccessful clinical case of aortic arch resection. They recognized that the small caliber

of the shunts they employed imposed an overwheling work load on the heart, and contributed to a prohibitive mortality. This was overcome by impeding the inflow of blood into the heart by means of partial occlusion of the inferior vena cava. The maneuver would seem only to add to the complexity of the problem. A simpler solution seems to be the enlargement of the caliber of the shunt.

In selecting the caliber of the shunt to be employed, the closer it approximates the lumen of the vessel to be by-passed, the fewer physiologic alterations will result. Izant, Hubay, and Holden[18] have shown experimentally in dogs that a shunt with a 5 mm. inside diameter will preserve 80 per cent of the blood flow and adequate pressure to prevent ischemic changes in the spinal cord and kidneys.[19] We have found in dogs that the use of a comparable shunt (6 mm. inside diameter) is associated with significant increases in pressure proximal to the shunt which drop to negligible levels if a 10 mm. inside diameter shunt is used.[20]

The optimum size of the shunt for clinical use is not known. The results of the pressure studies in Case 2 and studies on dogs indicates that approximately 10 mm. should be the minimum inside diameter. In older subjects with varying degrees of coronary artery disease and hypertension, the increase in cardiac work imposed by shunts of smaller caliber would not be well tolerated.

SUMMARY AND CONCLUSIONS

Two cases of aneurysm of the intrathoracic aorta treated by surgical excision and graft replacement, employing an external shunt of large caliber during the period of aortic dissection and occlusion, are presented.

The first case was a fusiform and saccular syphilitic aneurysm of the entire intrathoracic aorta which was excised with restoration of the arch by freeze-dried aortic heterograft. The procedure was technically achieved but the patient expired during recovery from anesthesia as a result of hemorrhage from the pulmonary artery stump of an incidental pneumonectomy. A shunt with a 10 mm. inside diameter functioned well during a six-hour and forty-five minute period of aortic obstruction.

The second case is a classical posttraumatic saccular aneurysm of the first portion of the descending aorta accompanied by a patent ductus arteriosus, successfully treated by partial aortic resection, restoration of aortic continuity by freeze-dried aortic homograft, and division of the ductus. Aortic pressures taken during the operation of the shunt disclosed distal aortic pressures to be adequate for prevention of renal and spinal cord ischemia. It is estimated that an external shunt for the adult human aorta should have a minimum inside diameter of 10 mm. throughout.

The false preoperative estimate that the second case would be amenable to local excision and aortorrhaphy suggests that a shunt and suitable graft should always be available when a case of intrathoracic aneurysm is explored.

The shunt employed in the cases presented not only provides a conduit during the period of aortic obstruction but introduces a factor of safety while the aneurysm is being dissected.

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(For Discussion, see page 102.)

RESECTION OF THE THORACIC AORTA WITH REPLACEMENT BY HOMOGRAFT FOR ANEURYSMS AND CONSTRICTIVE LESIONS

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ONE of the most significant advances in the field of cardiovascular surgery made during the past decade has been the development of the concept and technique of excisional therapy for certain congenital and acquired lesions of the aorta. The first successful resection of a segment of the aorta was reported by Gross[31] and Crafoord and Nylin[18] in 1945, both of whom excised a coarctation of the aorta and performed end-to-end anastomosis. In lesions where this can be accomplished, this procedure still provides the most effective method of therapy. On the other hand, in certain unusual types of coarctation, a lengthy segment of aorta must be removed, thus precluding the performance of end-to-end anastomosis. Various methods of dealing with such situations have been proposed but these also impose certain limitations upon operability. In some cases of atypical coarctation, for example, the problem may be solved by utilizing the left subclavian artery as a shunt to bridge the aortic defect by a method used experimentally by Blalock and Park.[8] In general, however, the problem of restoration of aortic function after extensive aortic resection required the development of a technique with wider applicability in order that more extensive resections could be satisfactorily accomplished. Aortic resection for lesions such as aneurysms not associated with vascular obstruction also imposed certain limiting factors upon the operation, control of which largely determines the success of aortectomy.

At the turn of the century Carrel[12] demonstrated experimentally that successful resection of the thoracic aorta in animals was possible and that the resulting defects in the aorta could be successfully repaired by vessel grafts. During the past decade the feasibility of aortic homografts used clinically to repair extensive surgical defects in the aorta created by resection of coarctation has been demonstrated by Gross,[32] and subsequently by others[2, 3, 9, 15, 21-24, 27, 28, 39, 41, 46] who have utilized the procedure in the treatment of aneurysms and constrictive lesions located in both the thoracic and abdominal aorta. The present limitations on the applicability of aortic resection include certain technical and physiological factors among which are the nature and extent of the lesion and its location. The purpose of this presentation is to consider these factors and the means of combating their limiting influence in a series of 12 aortic

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Aided in part by a grant from the Houston District Chapter, Texas Heart Association.

Read at the Thirty-fourth Annual Meeting of The American Association for Thoracic Surgery at Montreal, Quebec, May 3 to 5, 1954.

resections and homograft replacements, 7 of which were done for aneurysms and 5 for constrictive lesions. (See Addendum.) In some cases grafts preserved in a modified Tyrode's solution were employed for this purpose, but in the majority of instances freeze-dried or lyophilized grafts were used. The criteria of selection and method of procurement, preservation, and final restoration of the graft for use are reported elsewhere.[20]

ANEURYSMS

Excisional therapy of thoracic aortic aneurysms was suggested and tried unsuccessfully by Tuffier[47] more than fifty years ago. In 1910, Carrel[12] stated: "The diagnosis of the aneurisms of the aorta can be made today at a comparatively early period. It is rational to think that some of them can be extirpated. Therefore we must find methods which permit repair to the aortic wall after a partial or complete resection." Yet not until two years ago was extirpation of aortic aneurysms first advocated as the procedure of choice in all cases of thoracic aneurysm where factors permitted its satisfactory application.[14] The inevitably fatal nature of aortic aneurysms justifies an aggressive approach to such lesions directed toward removal of the irreparably damaged tissue and restoration of aortic continuity.

In general, two types of procedures may be employed for this purpose, namely, tangential excision with lateral aortorrhaphy, and resection of the involved segment of aorta with replacement by aortic homograft. For sacciform lesions involving the ascending aorta and proximal portion of the aortic arch the former procedure may be used. By placing a clamp across the neck of a sacciform aneurysm in the proximal regions of the aorta, the sac distal to the clamp may be excised and the aorta repaired without encroachment upon its lumen. This is obviously an important practical method of dealing with sacciform lesions in the ascending aorta and arch since it precludes the necessity of temporary arrest of the circulation, which, at a high aortic level, could easily produce lethal ischemic damage to the brain as well as left ventricular dilatation and fatal cardiac failure.

Fusiform aneurysms of the thoracic aorta, which involve the entire circumference of the vessel, present a special problem in that extirpation requires removal of an entire segment of the aorta. Aneurysmectomy for such lesions presents two problems; one is concerned with the ischemic effect on tissues distal to the point of aortic occlusion, and the other with bridging the defect created by aortic resection. The latter appears to have been satisfactorily solved for the present by the use of preserved aortic homografts. Thus, solution of the former problem represents the critical factor in successful performance of the operative procedure. A number of factors may influence the degree of ischemic damage occurring during the period of temporary interruption of aortic circulation. The level of occlusion is obviously significant. Even brief periods of occlusion of the ascending aorta or the transverse arch of the aorta are likely to be fatal. Thus, for the present, lesions in this region remain inoperable by clinically proved methods. On the other hand, temporary occlusion of the thoracic aorta distal to the left carotid or left subclavian artery may be tolerated by the myocardium if certain undesirable sequelae from

ischemia to the tissues distal to the occlusion can be controlled satisfactorily. Experience indicates that the spinal cord is the most vulnerable tissue to temporary aortic occlusion at these high levels. The degree of renal ischemia

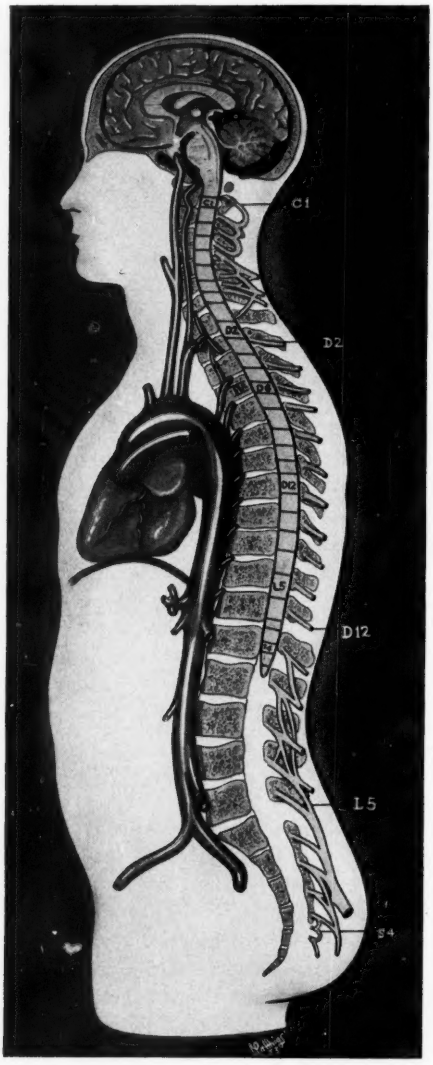


Fig. 1.—Diagrammatic drawing of sagittal section of body showing relationship between the anatomic levels of aorta, vertebral column, and spinal cord.

produced by temporary aortic occlusion above the renal arteries has not resulted in significant impairment of renal function, in our experience, nor has

there been any evidence of ischemic damage to other abdominal viscera or the lower extremities. Thus, prevention of spinal cord damage during occlusion of the descending thoracic aorta is usually the critical factor in the success of the procedure.

In general, the level and the duration of occlusion of the aorta determine the extent of cord damage. The distal portion of the aortic arch is located approximately at the level of the fourth thoracic vertebra and the origin of the sixth dorsal nerve from the spinal cord, and the aortic hiatus in the diaphragm is approximately at the level of the fifth lumbar nerve (Fig. 1). The lower end of the spinal cord is located opposite the first or second lumbar vertebra. Whereas cross-clamping the thoracic aorta distal to the arch may produce a severe paraplegia or even death from spinal shock, occlusion of the abdominal aorta below the renal arteries, as in resection of arteriosclerotic aneurysms in this region, rarely is complicated by spinal cord damage. Thus, in 45 cases of abdominal aneurysms in which the aorta distal to the renal arteries was occluded for periods up to two hours, there has not been a single instance of neurologic change in the lower extremities after operation.

Another factor influencing the degree of ischemic damage to the spinal cord is the necessity for double-clamping the aorta above and below the lesion in order to excise the intervening segment. Experimental evidence indicates that cross-clamping at two sites simultaneously, one on the proximal and the other on the distal thoracic aorta, increases the paraplegia and mortality rates significantly in animals.[42] Furthermore, in aneurysmectomy the risk is increased by the necessity for division of varying numbers of intercostal arteries, depending upon the extent of the lesion. In the experimental animal, division of intercostal arteries concomitantly with temporary aortic cross-clamping increases the incidence of paraplegia as well as the mortality. Thus it would appear that the length of the lesion influences significantly the risk of excisional therapy, since this necessitates division of increasing numbers of intercostal arteries.

Still another important consideration is the extent to which collateral circulation develops around the lesion. In instances of insidious or chronic occlusion of the aorta as in coaretation, the rich collateral network which is usually present insures against serious ischemic damage occurring even during prolonged periods of aortic cross-clamping. Consequently, few cases of paraplegia resulting from resection of the aorta for coaretation have been reported.[5, 7, 33] Conceivably collateral circulation may be mildly stimulated by the presence of an aortic aneurysm, and tissues below the lesion may even develop a relative tolerance to chronic partial ischemia. Although theoretically these factors may exert some protective influence, their clinical effectiveness in preventing ischemic changes during temporary aortic occlusion cannot be relied upon.

Still another factor influencing the degree of ischemic damage produced by arrest of aortic circulation is the duration of occlusion. The critical safe period of aortic occlusion has not been accurately determined, being quite

variable in the reported experience. Bahnson[2] reported cross-clamping the distal portion of the aortic arch for 7 minutes in one case and 20 minutes in another without evidence of ischemic cord damage, and Crafoord[19] reported, in a case of patent ductus arteriosus, occluding the aorta for 27 minutes without paralysis. Our experience indicates that the safe period may extend even

TABLE IA. RESECTIONS OF THE THORACIC AORTA WITH REPLACEMENT BY HOMOGRAFT FOR ANEURYSMS

Case	Blood Pressure	Lesion	Etiology	Date of Operation	Method of Repair	Result	
						Functional	Blood Pressure
8. E.L. 35yr. W♂	LA 260/160 LL 0/0		Acquired Arteriosclerotic	6-16-53		Fair	LA 160/110 LL 110/90
E.L. 35yr. W♂	LA 160/110 LL 110/90		Same 2nd operation	10-26-53		Excellent	LA 140/90 RL 170/100
9. S.S. 12 yr. W♂	RA 155/90 LA 110/90 LL 0/0		Congenital	8-17-53		Excellent	RA 124/60 LA 100/60 LL 130/70
10. A.T. 20yr. W♂	RA 190/110 RL 0/0		Congenital	9-15-53		Excellent	RA 140/90 RL 170/100
11. J.H. 30yr. W♂	RA 190/110 LA 130/140 RL 0/0		Congenital	10-27-53		Excellent	RA 150/90 LA 120/90 RL 130/100
12. J.O'G. 29yr. W♀	RA 160/90 LA 160/110 RL 0/0		Congenital	3-26-54		Excellent	RA 150/90 LA 146/90 RL 150/110

longer (Table IA). Nevertheless, the duration of occlusion in operations for thoracic aortic aneurysms must be minimized regardless of the factors which may tend to prolong this critical safe period.

Accordingly, certain technical steps in the operation designed to minimize the period of aortic occlusion assume practical importance. In general the method which we have used in all cases has been similar to that employed in Case 2 which will be used as an illustrative example (Figs. 2, 3). Adequate exposure is achieved by posterolateral thoracotomy; one usually resects a rib for this purpose. Where a long segment of aorta must be visualized, more satisfactory exposure is obtained by dividing the posterior ends of several adjacent ribs. In an occasional instance, as in Case 6 in which the entire thoracic aorta is involved, it may be practical to make a double entry into the pleural space by opening both the fourth and the eighth intercostal spaces in order to expose adequately the proximal and distal ends of the lesion (Fig. 4). All preliminary dissection and mobilization of the adjoining aorta are completed, and temporary encircling tapes are applied. After resectability of the lesion is determined, the graft is prepared for use.[20]

With everything in readiness the proximal and distal clamps are applied, and the exact time of occlusion is noted. Then the aneurysm is quickly removed, larger bleeders being ligated as they are divided. In extensive lesions, total removal of the aneurysm at this moment would involve a tedious dissection with possible damage to adjacent organs, thus delaying the ultimate restoration of aortic circulation. Under such circumstances only enough of the aneurysm is removed initially to permit trimming of the proximal and distal ends of a normal aorta and to provide a trough for placement of the graft (Fig. 3). Complete evacuation of the aneurysm and excision of the sac are postponed until after the graft has been inserted and aortic flow has been resumed. In performing the anastomosis, considerable time may be saved by utilizing a simple over-and-over continuous suture using 0000 arterial silk interrupting the suture line at two or three points along the circumference of the anastomosis. Upon opening the clamps there may be temporary bleeding from needle holes, but this will cease usually after a few minutes. Occasionally additional sutures may be required in the anastomosis. When aortic continuity is completely restored, the remainder of the sac is removed. If erosion of the vertebral bodies has occurred, there is ordinarily no sac present posteriorly, and the laminated thrombus is in contact with the marrow cavity of vertebrae. In some instances where the graft is suspended across such a large excavation, it is advisable to wrap the graft either with some adjacent pleura or else to transplant into the thorax a pedicled flap of omentum which may be brought through the aortic hiatus in the diaphragm. Aortic occlusion time in the chest should not exceed one hour and in less extensive lesions may be approximately thirty minutes. Anticoagulants are not used during or after such operative procedures.

In addition to minimizing the duration of aortic occlusion, other possible solutions to this problem include the use of shunts around the occluded seg-

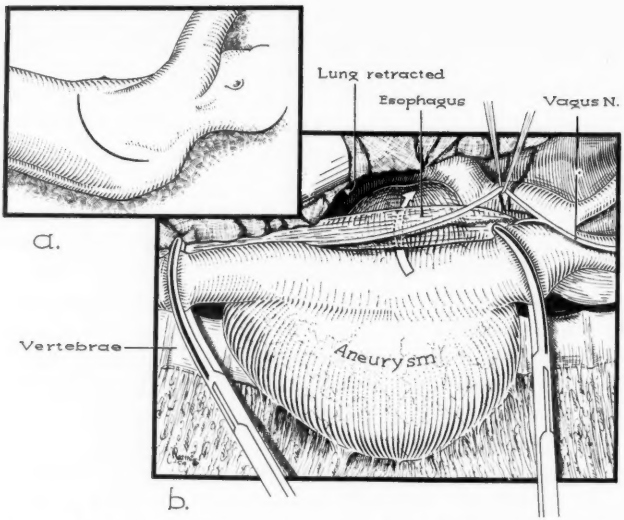


Fig. 2.—Drawing showing operative technique employed in Case 2 used as illustrative example of procedure usually employed in resection of descending thoracic aorta for aneurysm. *a*, Left posterolateral thoracotomy incision is employed dividing posterior ends of adjacent ribs. *b*, Aneurysm is demonstrated in relationship to vertebrae posteriorly and esophagus and lung anteriorly. Arrow indicates retroesophageal projection of aneurysm to the right.

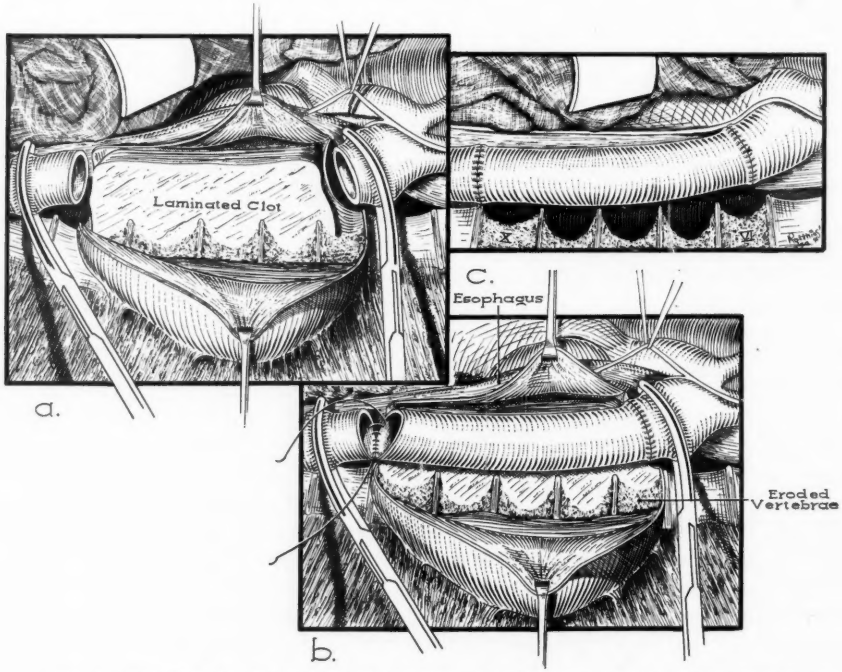


Fig. 3.—Drawing showing method of insertion of aortic homograft to restore circulatory continuity. *a*, After clamping aorta above and below lesion, a portion of the aneurysm is excised and the ends of the aorta trimmed. *b*, Graft lies in the lumen of the incompletely excised aneurysm as the anastomoses are performed with continuous over-and-over suture of 0000 arterial silk interrupted at several points. Upon restoration of circulatory continuity of aorta, remainder of aneurysm and contents are removed. *c*, Graft which bridges vertebral excavation is covered with adjacent pleura.

ment of aorta and the use of hypothermia. The former may be achieved by temporary intraluminal or external shunts.[13, 16, 26, 37, 38, 43] Both of these methods have been employed experimentally, and although they would appear to be quite useful for this purpose, their clinical effectiveness has not been demonstrated. Another possible solution to this problem lies in the use of hypothermia in order to reduce the arterial oxygen requirements of the central nervous system and thus prolong the period of safe aortic occlusion.[6] The protective effect of hypothermia in preventing ischemic damage during experimental temporary occlusion of the aorta has been clearly demonstrated.[4, 42]

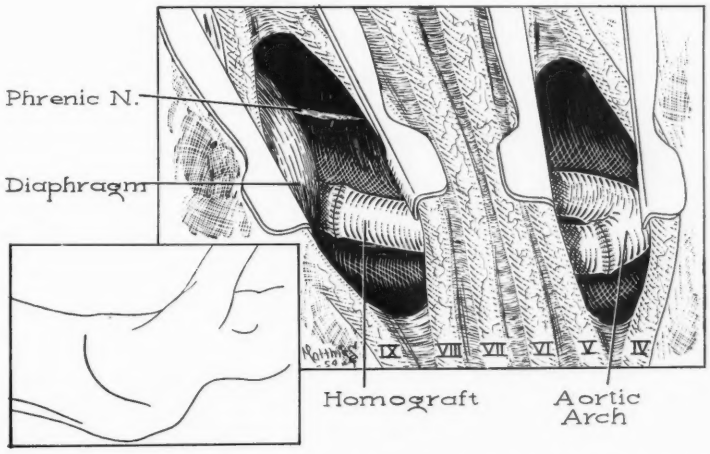


Fig. 4.—Drawing showing incision used in Case 6 where length of the lesion required exposure of entire descending aorta. Single incision is made through skin and soft tissues, and pleural cavity is opened through both the fourth and eighth intercostal spaces facilitating performance of the aneurysmectomy and anastomoses.

In our own experiments with temporary aortic occlusion, the mortality in 50 control animals was 32 per cent and in a similar number of hypothermic animals, 25 per cent. In these same experiments the rate of paraplegia in the surviving controls was 65 per cent, and in the hypothermic group no instance of paraplegia was encountered. From these observations it may be concluded that hypothermia deserves cautious clinical trial, particularly with operations for the more proximally situated aneurysms of the descending thoracic aorta. Local cooling of only the spinal cord proper by local application of cold packs to the spinal column may reduce both the temperature of the cerebrospinal fluid and the oxygen requirements in the spinal cord. An advantage of the latter technique is that some of the systemic factors may be eliminated which make general hypothermia somewhat dangerous, particularly when used in patients of advanced years, but further experience will be necessary before spinal cooling can be recommended for this purpose. We have used general hypothermia twice (Cases 5 and 6) and local or spinal hypothermia once (Case 7),

and although the thoracic aorta was occluded for 53, 62, and 53 minutes, respectively, in none of these was there evidence of paraplegia after operation.

The following case reports of our experience with resection of aneurysms of the thoracic aorta emphasize certain anatomic and physiologic considerations encountered in the excisional therapy of aortic aneurysms and represent a consecutive series of 7 patients treated by aneurysmectomy and aortic homograft.

CASE REPORTS (ANEURYSMS)

CASE 1.[21]—R. A., a 46-year-old white man, was admitted to Methodist Hospital on Dec. 31, 1952, with excruciating pain of three months' duration located in the lumbar region of the back, abdomen, and inguinal region, requiring opiates for relief. Palpation of the left costovertebral angle elicited pain, and there was roentgenographic evidence of an aneurysm of the lower thoracic and upper abdominal aorta producing erosion of the bodies of the ninth, tenth, eleventh, and twelfth thoracic and the first lumbar vertebrae. Serologic test for syphilis was positive.

On Jan. 3, 1953, through a left thoracoabdominal incision, the aneurysm was resected, and the 15 cm. defect in the aorta was repaired with a homograft preserved in modified Tyrode solution (Table IA). The period of aortic occlusion was 45 minutes, and there were no neurologic sequelae after operation. Convalescence was uneventful and he was discharged from the hospital thirty-one days later.

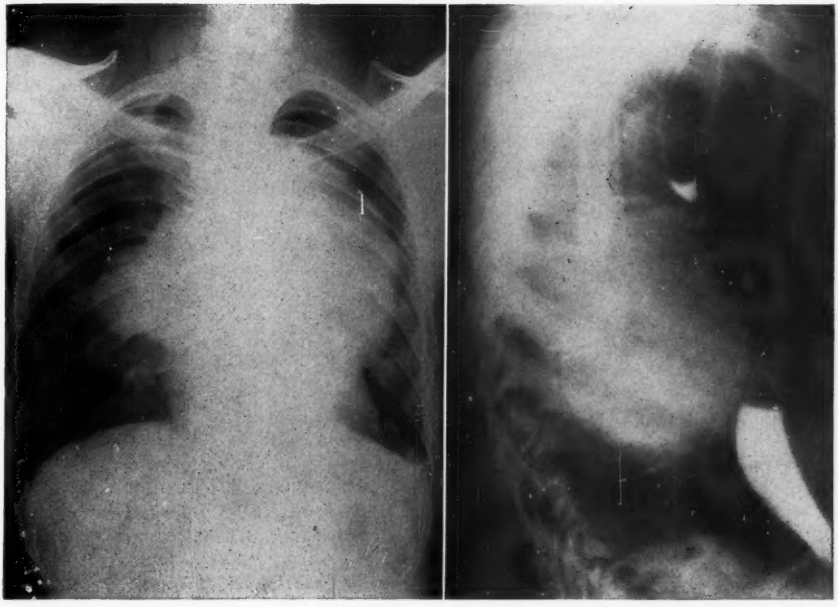
The patient has remained well and free of pain and has returned to work. Recent roentgenograms of the thoracolumbar spine reveal progressive regeneration of the eroded vertebral bodies, and there is no evidence of dilatation of the graft.

Comment.—To our knowledge this is the first successful resection of an aneurysm of the thoracic aorta with homografting. Lam and Aram[39] previously reported a similar case in which a large part of the aneurysmal sac and contained thrombus were not removed, resulting in infection several months later with fatal secondary hemorrhage. It is important, as emphasized previously,[2, 14, 15, 39] to extirpate as much of the aneurysm and thrombus as possible.

The homograft which lay suspended across the large vertebral excavation in our Case 1 lacked soft tissue support so this was supplied by utilizing the pedicled omentum to wrap the graft. Follow-up period in this case is approximately sixteen months, and the graft shows no evidence of degeneration.

CASE 2.—O. J., a 50-year-old Negro man, was admitted to Jefferson Davis Hospital, June 3, 1953, with excruciating pain in the left chest requiring opiates for relief. In 1942 he had experienced substernal pain radiating to the back, and in 1947 he was admitted to the hospital because of severe pain in the left chest following the course of the fifth and sixth intercostal nerves. On that admission, roentgenogram of the chest revealed an aortic aneurysm and destruction of the thoracic verebral bodies. Serologic tests for syphilis were positive, and the patient received 6 million units of penicillin. Prior to the present admission he had been observed on several occasions in the medical clinic for the aneurysm, and during this period he was almost totally disabled by pain in the back and left chest.

Physical examination revealed a thin, chronically ill appearing man in constant discomfort. There were normal carotid and brachial pulsations and blood pressure was 140/90 mm. Hg. The heart was mildly enlarged, and there was no evidence of aortic insufficiency. There was dullness to percussion over the posterior lung fields, and breath sounds were absent in this area. There was a palpable pulsation in the posterior chest wall near the



A. *B.*

Fig. 5.—Roentgenograms of chest in Case 2 demonstrating in the posteroanterior projection (*A*) an extensive aneurysm of the descending thoracic aorta presenting both to left and right of mediastinal shadow, and in the lateral (*B*) the retroesophageal position of lesion displacing esophagus and trachea forward.

Fig. 6.—Oblique roentgenogram of the chest in Case 2 showing striking distortion of esophagus and extensive erosion of bodies of sixth through tenth thoracic vertebrae.

inferior angle of the left scapula. The remainder of the examination was not remarkable. Hemogram and urinalysis were within normal limits, and serologic tests for syphilis were negative.

Roentgenograms of the chest revealed a large mass in the posterior mediastinum extending to the left within another process extending to the right (Fig. 5). Esophagrams showed a distortion of the esophagus with displacement to the left (Fig. 6). There was erosion of the bodies of the sixth through the tenth thoracic vertebrae. Fluoroscopy revealed minimal pulsation in the mass.

On June 11, 1953, a left thoracotomy was performed through the bed of the resected seventh rib, and to obtain adequate exposure the posterior ends of the fifth, sixth, and eighth ribs were divided. The aneurysm was extensive and was densely adherent to the posterior surfaces of the left upper and lower lobes of the lung. Proximally, the aneurysm arose about 1.5 cm. distal to the left subclavian artery and extended distally to a point about 5 cm. above the aortic hiatus in the diaphragm (Table IA). Posteriorly, there was no definite sac, and there was a deep excavation of the vertebral bodies leaving the intervertebral bodies relatively uninvolved. There was an extension of the sac far to the right of the midline and posterior to the heart, and this was encroaching upon the ribs in the right costovertebral gutter. The aneurysm, together with the contained laminated thrombus, was completely removed, and a 23 cm. homograft preserved in modified Tyrode solution was inserted (Figs. 2 and 3). The graft preserved in modified Tyrode solution was inserted (Figs. 2 and 3). The period of aortic occlusion was 45 minutes. At the completion of the procedure circulation to the lower half of the body was satisfactory. A prophylactic tracheostomy was performed.

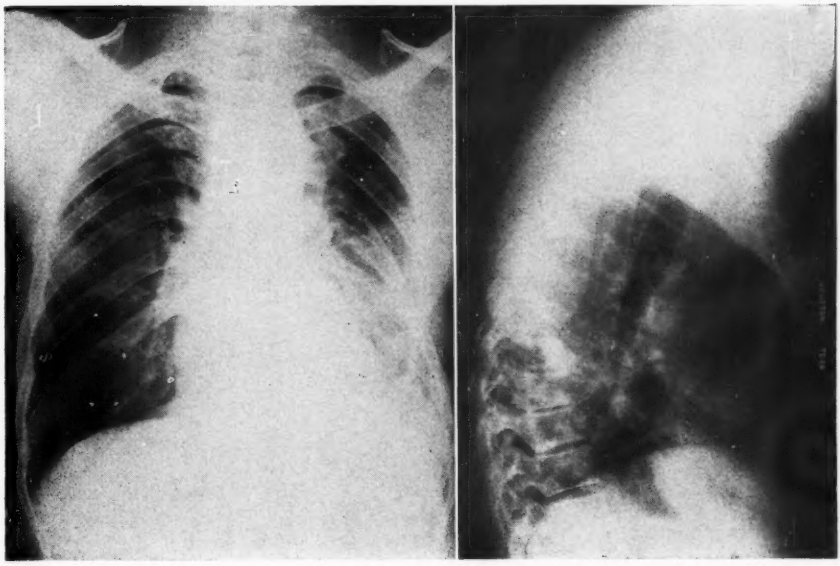
Urinary output during the first twenty-four hours after operation was 750 c.c., and no renal complications ensued. His general condition improved steadily, and after about ten days he was completely free of pain and his appetite returned. On the second day postoperatively it was noted that there was diminished sensation in the feet and general weakness of all muscles of the lower extremities. This weakness never developed into frank paralysis, and he had no loss of sphincter control of the anus or bladder. After intensive physical therapy he left the hospital ambulatory on Aug. 17, 1953, using a cane for support.

Since dismissal from the hospital, the patient has improved progressively, and when last examined on April 15, 1954, he had gained 30 pounds and walked unassisted with minimal residual weakness in his legs. Roentgenograms of the chest at this time showed progressive resolution of the residual pleural thickening on the left. There was kyphoscoliosis in the region of the vertebral erosion, but callus formation indicated satisfactory healing (Figs. 7 and 8).

Comment.—In spite of the magnitude of the lesion in this case, successful surgical correction was achieved, and almost the entire descending thoracic aorta was replaced by a homograft measuring 23 cm. in length. The neurologic sequelae, although troublesome, were not disabling, and the patient has been completely rehabilitated. The result in terms of palliation is striking, and life expectancy has unquestionably been extended.

CASE 3.—T. M., a 58-year-old man, was admitted to Jefferson Davis Hospital on Nov. 10, 1953, complaining of intermittent, severe pain in the left lower chest posteriorly radiating to the epigastrium. He was in good health until he sustained rib fractures on the right in an automobile accident in June, 1953.

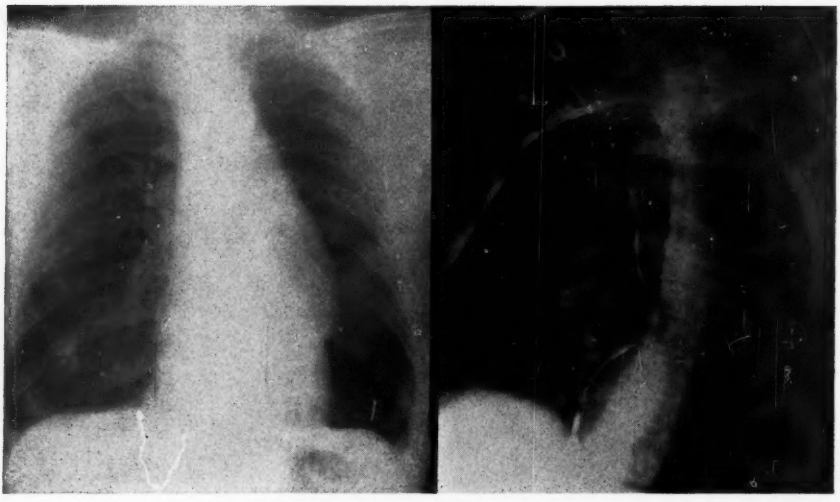
He was in constant discomfort and appeared chronically ill. Blood pressure was 138/90 in both arms, and the heart was slightly enlarged with a Grade II aortic systolic murmur. There was dullness to percussion over the left chest posteriorly but no abnormal pulsations.



A. *B.*

Fig. 7.—*A* and *B*, Roentgenograms of the chest in posteroanterior and lateral projections in Case 2 made ten months after operation, demonstrating absence of aneurysm and residual pleural thickening on left.

Fig. 8.—Oblique roentgenogram of thoracic vertebrae in Case 2 made ten months after operation showing moderate kyphoscoliosis and progressive healing with callus formation in the involved vertebrae.



A. *B.*

Fig. 9.—Roentgenograms of chest in Case 3 showing in posteroanterior projection (*A*) a tortuous fusiform aneurysm of the entire descending thoracic aorta, and in the oblique (*B*) deviation of the esophagus by aortic elongation and kinking.

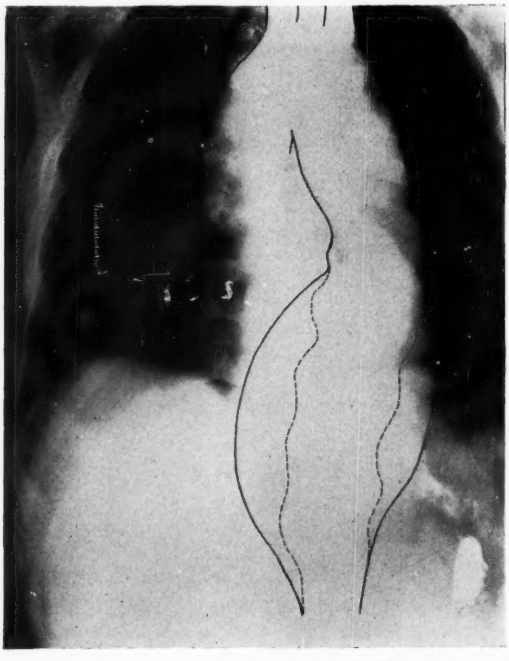


Fig. 10.—Retouched retrograde aortogram in Case 3 demonstrating elongated aorta and fusiform aneurysm with distal fusosacciform dilatation containing laminated thrombus.

Serologic test for syphilis was positive. Electrocardiogram revealed left ventricular hypertrophy and suggested anterolateral ischemia.

Roentgenograms of the chest showed moderate dilatation of the ascending aorta and an extensive aneurysm involving the entire descending thoracic aorta (Fig. 9). These findings were confirmed by a retrograde aortogram (Fig. 10).

He received 2,400,000 units of penicillin daily for five days prior to surgery. Following retrograde aortography he was hypotensive for twenty-four hours, probably due to impaired myocardial function.

Thoracotomy was performed Nov. 9, 1953, under ether anesthesia. A single posterolateral incision was made through soft tissues, but in order to expose the aorta proximal and distal to the aneurysm, the pleural cavity was entered in both fourth and eighth interspaces (Fig. 4). The fusiform aneurysm extended from the level of the left subclavian

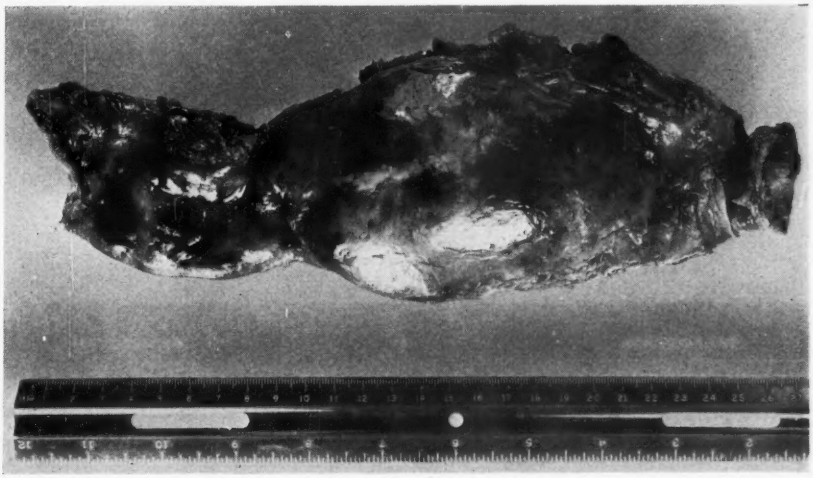


Fig. 11.—Photograph of specimen removed in Case 3 showing aneurysm involving entire descending thoracic aorta.

artery proximally to the level of the aortic hiatus distally (Table IA). The upper portion of the aneurysm was not as large as the distal portion where the lesion was approximately 12 cm. in diameter and was eroding the vertebrae (Fig. 11). The aorta was clamped above and below the aneurysm for 77 minutes while a segment of aorta measuring 30 cm. in length was resected and replaced by a lyophilized homograft. Operation lasted 4 hours and 45 minutes, and he received a 3,500 c.c. blood transfusion.

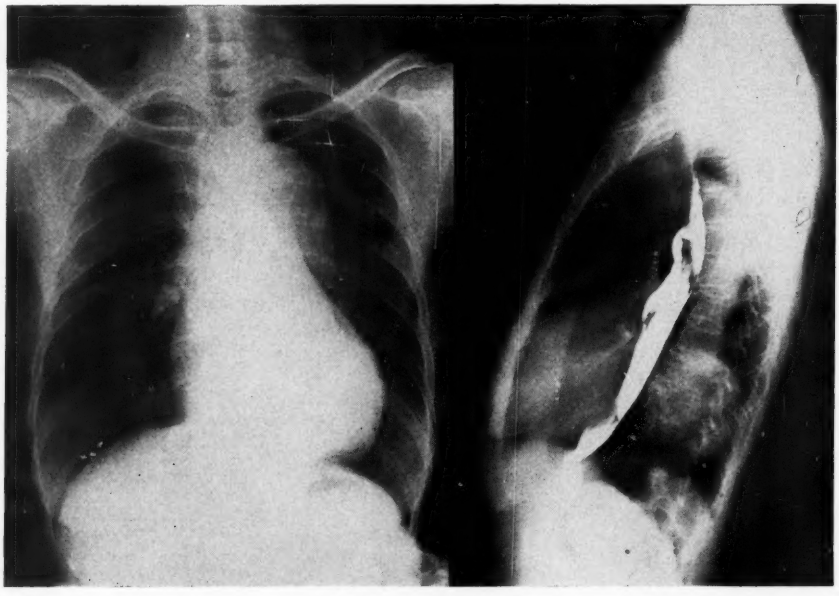
During the postoperative period a constant drip of Levophed (nor-epinephrine) was necessary to maintain blood pressure at 9-100/50-60 mm. Hg. He moved his lower extremities well, and urinary excretion was satisfactory. About twelve hours after operation Levophed was inadvisedly discontinued, and before it could be restarted the patient became restless, suddenly hypotensive, and expired.

Autopsy revealed that the aortic homograft was intact and presumably functioning satisfactorily. The heart showed no evidence of recent infarction although there was coronary arteriosclerosis and histologic evidence of myocardial fibrosis.

Comment.—In this case the aortic homograft, measuring 30 cm. in length, was the longest used in our series. The entire descending aorta was removed from just below the left subclavian artery to the aortic hiatus in the diaphragm

dividing all of the intercostal arteries. In spite of this extensive aortic resection the patient moved his lower extremities well following operation. Adequate exposure was obtained by utilizing the technique of double entry into the chest by separate intercostal incision. The decision to remove the entire aneurysm, involving the entire descending thoracic aorta, was probably a mistake, and only the distal portion of the aneurysm which was eroding vertebrae and causing severe pain should have been removed. Obviously, removal of the entire lesion increased the operative risk.

CASE 4.—E. M., a 50-year-old Negro woman, was admitted to Jefferson Davis Hospital on Nov. 12, 1953, with severe pain in the left chest of eighteen months' duration. The



A. *B.*

Fig. 12.—*A* and *B*, Roentgenograms of the chest in Case 4 in posteroanterior and lateral projections showing fusiform dilatation of the entire thoracic aorta with fusosacciform protuberance in the distal half of the descending aorta.

pain originated posteriorly near the inferior angle of the scapula and radiated obliquely anteriorly and inferiorly. Opiates were necessary for relief of pain. She had lost 20 pounds in weight.

Physical examination revealed a poorly developed and chronically ill appearing woman who was in moderate discomfort. The blood pressure was 170/100 mm. Hg. The heart was enlarged, and there was an aortic systolic murmur Grade II, but no evidence of aortic insufficiency. The remainder of the examination, including a neurologic survey, was not remarkable.

The serologic test for syphilis was positive, but the remainder of the laboratory studies were considered normal.

Roentgenograms of the chest revealed moderate cardiac enlargement, mostly left ventricular (Fig. 12). The entire aortic arch was involved in a fusiform aneurysmal dilata-

tion. In the distal half of the descending thoracic aorta, there was a fusosacciform aneurysm occupying the costovertebral sulcus posteriorly but demonstrating no erosion of the vertebrae or ribs.

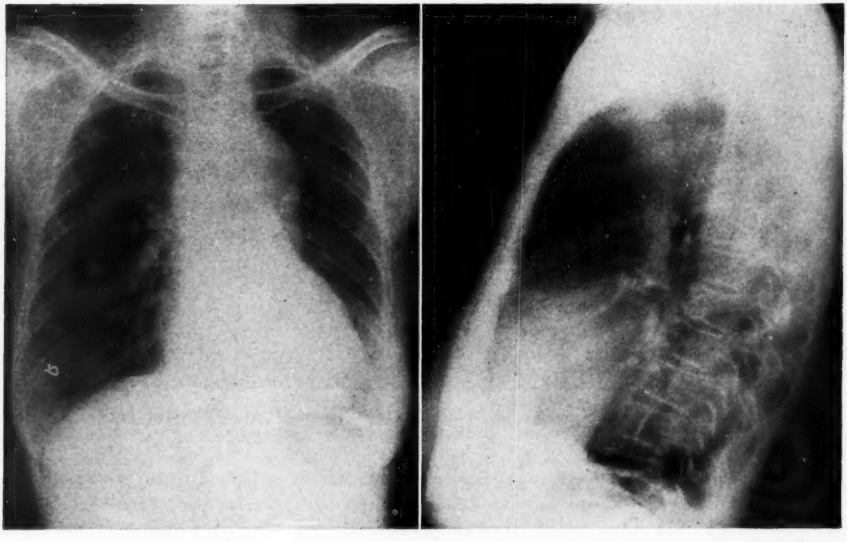
On Nov. 19, 1953, under ether anesthesia a left posterolateral thoracotomy was done through the seventh intercostal space. There was a large fusiform dilatation of the entire aortic arch and proximal descending aorta (Table IA). The aneurysm in the lower thoracic aorta was fusosacciform and extended over a distance of approximately 10 cm. The aortic diameter proximal to the lesion was about twice the size of the distal aorta, and the lesion extended to the diaphragm below (Fig. 13). Because of the distribution of the pain, only the aneurysm of the lower thoracic aorta was resected. The lesion was

Fig. 13.—Photograph of specimen removed in Case 4 showing fusosacciform aneurysm with discrepancy in diameter of the aorta proximal and distal to lesion.

pressing on the vertebrae and ribs posteriorly, but erosion of bony structures was absent. Continuity of the aorta was re-established by inserting a 12 cm. lyophilized segment of thoracic aorta (Fig. 14). When the clamps were removed, 38 minutes after they had been applied, circulation to the lower half of the body was re-established.

Her postoperative course was smooth, with no evidence of any neurologic disturbances, and she was discharged from the hospital on Dec. 13, 1953. When last seen in

Fig. 14.—Photograph made at operation in Case 4 showing 12 cm. homograft inserted into aortic defect. Lower anastomosis is located about 5 cm. above aortic hiatus in diaphragm.

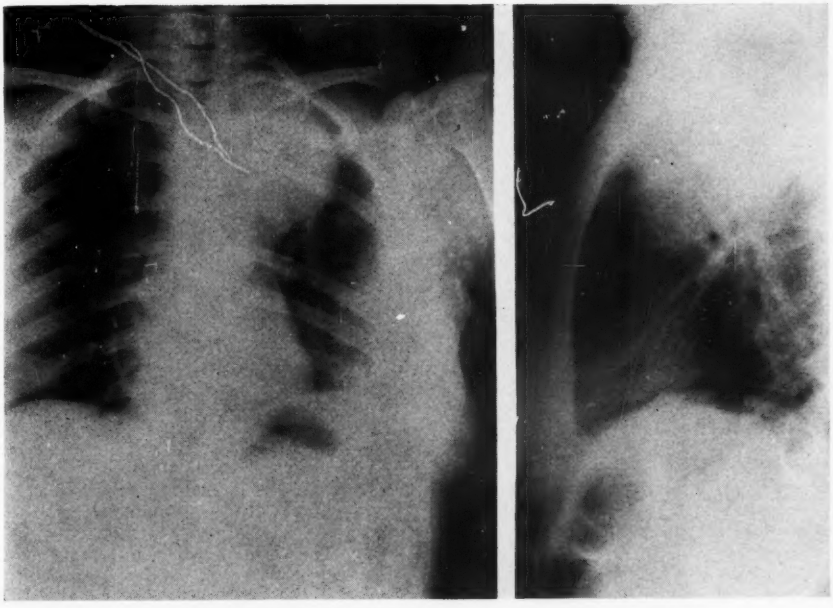


A. *B.*

Fig. 15.—*A* and *B*, Posteroanterior and lateral roentgenograms of chest in Case 4 made five months after operation showing persistence of fusiform aneurysm of the aortic arch and absence of fusosacciform lesion of the descending thoracic aorta.

the chest clinic on April 6, 1954, she was free of pain and had gained 15 pounds. Roentgenograms of the chest revealed progressive clearing of the pleural thickening at the left base (Fig. 15), and fluoroscopy revealed no dilatation in the graft.

Comment.—In this case only the symptomatic portion of the aneurysm, i.e., the fusosacciform dilatation in the lower thoracic aorta, was resected. In contrast to Case 3 where the entire descending thoracic aorta was removed and death ensued, this patient has responded well. Apparently the sacciform protuberances on the aorta represent the site of maximum degeneration and weakness and are most likely to cause pain and ultimately to perforate.



A. *B.*

Fig. 16.—*A* and *B*, Posteroanterior and lateral roentgenograms of chest in Case 5 made six weeks after aneurysmectomy performed elsewhere showing large aneurysm of distal half of aortic arch.

CASE 5.—D. J., a 17-year-old white girl, was admitted to Methodist Hospital on Dec. 25, 1953, with a diagnosis of aneurysm of the aortic arch and suspected septicemia. She had been healthy and was attending public high school where a routine survey roentgenogram of the chest revealed a small cystic mass in the region of the left upper lobe. Exploratory thoracotomy was done on Nov. 11, 1953, at another hospital, at which time a small thin-walled aneurysm of the arch of the aorta measuring about 2.5 cm. in diameter was discovered arising just distal and adjacent to the ligamentum arteriosum. This lesion was excised tangentially, and aortorrhaphy was accomplished with considerable difficulty due to the thinness and friable nature of the neck of the lesion. After this operation she ran a febrile course, and septicemia was suspected but blood cultures were sterile. She was discharged from this hospital about three weeks later but readmitted in three weeks again with fever, 106° F., and one spell of convulsions. In spite of intensive antimicrobial treatment, fever persisted,

and she developed severe substernal pain and a "brassy" cough. Blood cultures were persistently sterile. Serial roentgenograms of the chest revealed an expanding mass in the upper left chest adjacent to the mediastinum.

She was then seen in consultation and transferred to Methodist Hospital. Roentgenograms of the chest upon admission revealed the mass to measure 8 by 9 cm. in diameter representing a rapid increase in size of the lesion during the previous twenty-four hours (Fig. 16). Laboratory studies revealed a moderate anemia but no other abnormalities.

Fig. 17.—Photograph made at operation in Case 5 showing aortic arch homograft inserted to restore aortic as well as left subclavian circulation.

Operation was performed on Dec. 25, 1953, under general anesthesia and hypothermia, which was induced in a special rubberized blanket containing coils through which a refrigerant solution was circulated. When the rectal temperature was reduced to 85° F., the blanket was removed and the operation was begun. Upon opening the previous thoracotomy incision it was apparent that the mass in the mediastinum was a large false aneurysm of the aorta intimately adherent to the left upper lobe (Table IA). The aortic arch was mobilized proximal to the lesion and the mid-thoracic aorta mobilized distally. A proximal clamp was applied across the arch of the aorta distal to the left common carotid artery, and a distal clamp was applied several centimeters below the lesion. Dissection of the aneurysm revealed that it had a false sac supported largely by the left lung. The aneurysm contained mostly fresh clots and originated from the site of the previous aortorrhaphy which had disrupted. A portion of left subclavian artery was excised with the aneurysm, and the defect in the arch

was replaced by a corresponding segment of lyophilized homograft of aortic arch measuring 7 cm. in length (Fig. 17). The patient's subclavian artery was anastomosed to the graft. Aortic occlusion time was 53 minutes, and the left subclavian was occluded an additional 12 minutes. The operation required 4 hours and 10 minutes. Rewarming of the patient was accomplished in the rubberized blanket, through which warm solution was circulated, and by use of heat cradles.

Her condition for the first three days was satisfactory. Urinary output was excellent, and there were no neurologic sequelae. However, oral temperatures steadily increased thereafter to a range of 101° to 103° F. and tachycardia persisted (130 to 160 beats per minute). At intervals she received penicillin, streptomycin, erythromycin, achromycin, Chloromycetin, and polymyxin in large doses without response. On the fifth postoperative day a positive blood culture for Klebsiella pneumoniae was finally obtained, and this organism was resistant to all antibiotics. Because of an increasing shadow in the chest films demonstrated at the left base, several unsuccessful thoracenteses were performed. In spite of repeated blood transfusions she expired on the eighth postoperative day.

Autopsy revealed multiple intrapulmonary abscesses and a hemopyothorax on the left. No definite source of bleeding was evident and the anastomoses were intact. There was also no evidence of infected vegetations in the graft or on the heart valves.

Comment.—General body hypothermia was used in this critically ill patient because the location of the aneurysm made it apparent that cross-clamping of the aortic arch would be required. It is significant, therefore, that in spite of the fact that the aorta was occluded just beyond the level of the left common carotid artery for almost one hour, no neurologic damage resulted. Indeed, the hypothermia appeared to play a vital part in increasing the patient's tolerance to an extensive operative procedure. It appears that she would have survived operation if the organism causing the septicemia could have been controlled. In this connection and in light of certain observations[10, 11] suggesting that tissue cooling may enhance infection, the hypothermic procedure in this case may have contributed to the difficulty in controlling the septic process.

CASE 6.[24]—W. M., a 31-year-old white man, was admitted to Methodist Hospital on July 5, 1953, with a history of having sustained a severe crushing injury to the chest in March, 1954, while on active duty in the Air Force. Hemothorax resulted which was successfully treated by thoracenteses, and since that time he was asymptomatic. Roentgenographic studies on admission revealed a partially calcified mass in the distal aortic arch. At thoracotomy on July 7, 1953, a fusiform aneurysm arising in the arch of the aorta just distal to the left subclavian artery and about 6 cm. long was identified. Partial aneurysmectomy was done excising tangentially the lateral projection of the lesion and aortorrhaphy was done with multiple interrupted silk sutures. Postoperative convalescence was uneventful and he was dismissed ten days later.

He remained well until December, 1953, when he complained of pain in the left chest, hoarseness, and difficulty in deep breathing. Roentgenograms of the chest suggested a recurrence of the aneurysm, and aortography confirmed the diagnosis. A second operation was done on Feb. 5, 1954, under general hypothermia. Body temperature was reduced gradually to 82° F. The aneurysm had recurred and enlarged so that it involved the entire aortic arch beginning at a point just distal to the left common carotid artery and extending about 8 cm. along the aortic arch and descending thoracic aorta distally (Table IA). The aorta was doubly clamped just distal to the left carotid artery and also distal to the aneurysm; and the lesion was resected, preserving the attachment of the subclavian artery. The 8 cm. defect in the aorta was restored with a comparable segment of aortic homograft preserved in modified Tyrode solution. The period of aortic occlusion

was 62 minutes. At the completion of the operation, which lasted 4 hours and 20 minutes, the patient's temperature was 83° F. Rewarming was accomplished with heat cradles and the special blanket circulating hot water.

About six hours after operation a massive hemothorax developed and reexploration and evacuation of the blood clots were necessary. A generalized ooze of blood from all raw surfaces was noted, and intravenous calcium gluconate, vitamin K, and protamine sulfate in addition to blood transfusions were used. The chest was closed with drainage. Recovery was complete, and he was discharged from the hospital fifteen days later. Since that time he has remained well and returned to work.

Comment.—To our knowledge, this is the first successful use of hypothermia for thoracic aneurysmectomy (See Addendum). Soon after the second operation there was evidence of a massive left hemothorax which required operative evacuation. There was an apparent temporary disturbance in the clotting mechanism producing a definite bleeding tendency after use of hypothermia. In this instance the bleeding tendency was apparently controlled by injection of calcium, vitamin K, and protamine sulfate, although the exact cause for the disturbance was not ascertained. The possible relationship of the hypothermic state to the clotting mechanism is not entirely clear.

CASE 7.—J. B., a 66-year-old Negro man, was admitted to Jefferson Davis Hospital Feb. 25, 1954, with severe aching pain in the left chest of six months' duration and a weight loss of 17 pounds. He had a history of a penile lesion fifteen years previously but received no treatment.

Physical examination revealed a blood pressure of 190/120. The trachea was deviated to the right, but there was no tracheal tug. The heart was slightly enlarged to the left, and there was a Grade II aortic systolic murmur and frequent premature beats. The left chest posteriorly was dull to percussion.

Serologic test for syphilis was positive. Electrocardiogram suggested anterolateral ischemia and left ventricular hypertrophy.

Roentgenograms of the chest showed a dilated ascending aorta and large fusosacciform aneurysm of the entire descending thoracic aorta (Fig. 18).

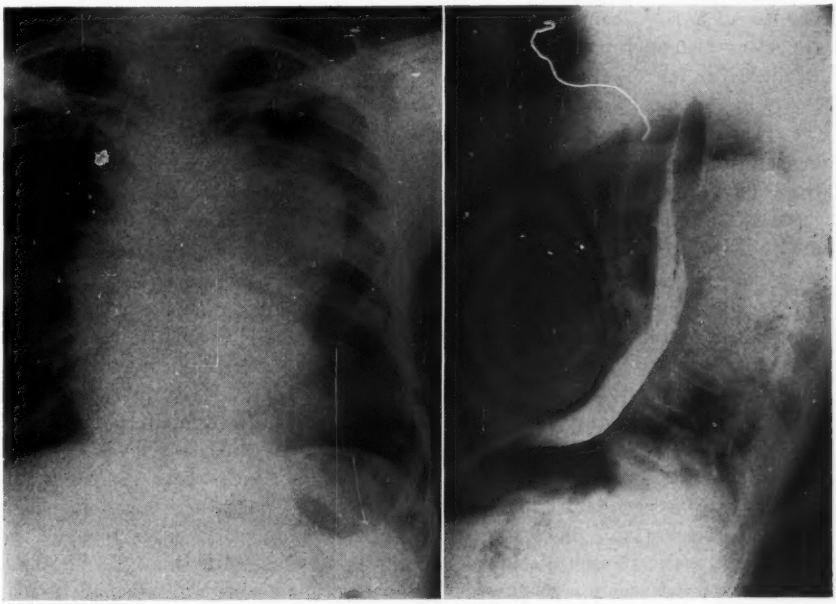
In spite of the fact that the patient was an obviously poor candidate for surgery, because of the disabling symptoms present operation was undertaken on March 4, 1954. Prior to operation local refrigeration of the spine by means of ice packs was employed for about three hours. During this interval the temperature of the spinal fluid decreased from 93.2° to 85° F. and body temperature decreased from 98.4° to 94.5° F. He described numbness in the feet and legs, but motor function was normal.

A left thoracotomy was performed resecting the sixth rib. The aneurysm arose about 3 cm. distal to the left subclavian artery and extended to a point 5 cm. proximal to the diaphragm (Table IA). The lung was adherent to the mass, and posteriorly the sac was absent, and the adjacent vertebrae and ribs were eroded. The defect in the aorta which measured about 18 cm. after the lesion was excised was bridged with a lyophilized aortic homograft. The aorta was occluded for 53 minutes. The operation lasted 3 hours and 25 minutes, and he received 3,000 c.c. blood transfusion. Blood pressure was 100/60, and his condition was fair at the conclusion of the procedure.

During the ensuing two hours he recovered from anesthesia and moved his lower extremities and talked to the attendants. About four hours after operation his blood pressure dropped to 80/50 in spite of intravenous Levophed (nor-epinephrine). A roentgenogram of the chest was obtained, and a massive left hemothorax was demonstrated. The patient's condition deteriorated rapidly, and while preparation was being made for operation he suddenly expired.

At autopsy the left chest contained an estimated 2,000 c.c. of liquid unclotted blood. No definite bleeding point was demonstrated, and the graft was intact. A generalized hemorrhagic ooze was the probable cause of the hemothorax. There was evidence of coronary sclerosis and focal myocardial fibrosis.

Comment.—In this elderly patient it was considered too risky to employ general body hypothermia; therefore, local application of cold to the spine was used to reduce the temperature of the cerebrospinal fluid. Although death occurred six hours after operation, he responded well enough in the postoperative interval to move his lower extremities voluntarily and appreciate painful stimuli. Whether or not the local hypothermia employed here had any influence on the subsequent bleeding tendency is debatable, but at the second thoracotomy it was evident that there was a capillary type of bleeding which was generalized.



A. *B.*

Fig. 18.—*A* and *B*, Posteroanterior and lateral roentgenograms of chest in Case 7 showing fusiform dilatation of aortic arch and fusosacciform aneurysm of proximal half of descending thoracic aorta.

CONSTRICTIVE LESIONS

Lesions producing an obstruction to circulation through the descending aorta may be either congenital or acquired. The usual congenital coarctation occurs in the isthmic region of the aortic arch where it joins the descending thoracic aorta just distal to the origin of the left subclavian artery and the ductus Botalli. In the typical coarctation only a short segment of aorta is

obstructed and local resection with end-to-end anastomosis is relatively simple and the procedure of choice. In these cases blood pressure in the carotid and subclavian arteries is elevated, but in the lower half of the body it is diminished and may be unobtainable. In the so-called "infantile" coarctation, the aortic constriction lies in a more proximal position, often above the left subclavian artery and usually involving a longer segment of aorta. Although in such cases death usually occurs shortly after birth, some patients survive and may live to adulthood. In those instances where the coarctation lies proximal to the left subclavian artery, there may be a striking discrepancy in the pulse and blood pressure obtainable in the two arms and hypertension in the right arm may be extremely high. The length of the constrictive segment in many of these cases together with its position makes resection with end-to-end anastomosis a virtual impossibility. Thus, in these unusual types of coarctation the procedure of aortic resection with restoration of circulation necessitates the use of aortic homografts in order to bridge the resulting defect. We are reporting two such cases (Cases 9 and 11) in which restoration of circulation through the aorta as well as through the left subclavian artery was achieved by employing a corresponding segment of aortic arch homograft.

In a significant number of cases aneurysms occur in the vicinity of the coarctation arising either from the aorta itself or from the point of origin of the dilated intercostal and bronchial arteries. In order to remove the pathologic constriction along with the secondary aneurysms, homografts should be available to bridge the defect.

Acquired constrictive lesions of the thoracic aorta are rare. In general they tend to occur farther along the course of the aorta than the usual site for congenital coarctations. There appears to be a localized aortitis resulting in segmental atherosclerosis producing the obliterative process in the aorta. The length and position of the process varies, but in the majority of cases which have been reported the involved segment measures approximately 4 to 6 cm. in length and is located in the middle or terminal thirds of the descending thoracic aorta (Table II). Only twelve such lesions have been reported, and in only six has surgical exploration been done. In the three cases in which surgical excision and placement of an aortic homograft were performed, the results were excellent. We are reporting a fourth case of this type in which there was a coarctation of the distal thoracic aorta and also a separate constriction in the upper abdominal aorta causing obliteration of the celiac axis (Case 8).

The operative procedure of resection with replacement by aortic homograft, essentially as done for aneurysms of the descending thoracic aorta, may also be employed for constrictive lesions. A favorable feature of the constrictive lesions in contrast to aneurysms is the presence of a well developed collateral network of vessels which provide adequate circulation to the tissues distal to the point of temporary cross-clamping and for almost indefinite periods of time. Thus, in these cases there is no need for haste in completing the resection and grafting after the clamps are applied. Furthermore, there

is no necessity for using shunts or hypothermia to protect distal tissues during periods of temporary occlusion. Emphasis should be placed on complete restoration of all major vascular channels, particularly the left subclavian artery when its origin is involved in the obliterative process. The following cases of aortic resection and replacement by homograft for constrictive lesions of the aorta are presented to illustrate some of the technical features of this problem.

TABLE II. REPORTED CASES OF COARCTATION OF THE THORACIC AORTA AT UNUSUAL SITES

AUTHOR	AGE (YR.)	SEX	LOCATION	SURGICAL TREATMENT	RESULT IN OPERATIVE CASES
1. Schlesinger (1835)[45]	15	F	Distal third	None	
2. Hasler (1911)[25]	49	M	Distal third	None	
3. Costa (1930)[17]	49	F	Middle third	None	
4. Hickl (1931)[36]	38	F	Middle third	None	
5. Hahn (1933)[34] Schleckat (1933)[44]	44	M	Distal third	None	
6. Olim (1949)[40]	20	F	Distal third	Aortic resection without repair	Died 5 hours later
7. Bahnson, et al. (1949)[1]	17	F	Middle third	Dorsal sympathectomy	Slightly improved
8. Freeman, et al. (1950)[29]	14	F	Distal third	None	
9. Beattie, et al. (1951)[3]	19	F	Distal third	Aortic resection, homograft	Excellent
10. Glenn, et al. (1952)[30]	19	F	Distal third and upper abdominal	By-pass using splenic artery	Good
11. Brock, Graham (1952)[9]	28	M	Distal third; aneurysm located proximal to stenosis	Aortic resection, homograft	Excellent
12. Deterling (1953)[27]	23	F	Distal third; obliterated left subclavian artery	Aortic resection, homograft	Excellent
13. Cooley, DeBakey (1954) (Case 8)	33	M	Distal third, proximla abdominal	Aortic resection, homograft	Excellent

CASE REPORTS (CONSTRICTIVE LESIONS)

CASE 8.—E. L., a 35-year-old white man, was admitted to Methodist Hospital on June 14, 1953, with a history of hypertension known to be present since 1939. He was rejected for military service on account of hypertension but had continued to work as a boilermaker. On April 7, 1953, while at work he was seized with a sudden headache and lost consciousness. Systolic blood pressure at that time was more than 300 mm. Hg. He developed a complete left hemiplegia which gradually improved, and at the time of admission he had slurring of speech and left hemiparesis. Because of religious beliefs he refused to receive a blood transfusion in any form.

Physical examination revealed an underdeveloped chronically ill appearing man who was apparently emotionally unstable. Blood pressure in the left arm was 260/160 mm. Hg and left leg 0/0. The heart was not enlarged, and there were no murmurs, although A_2 was accentuated. Moderate development of collateral vessels was present in the chest wall, and a systolic bruit was audible over the mid-thoracic spine to the left of the midline.

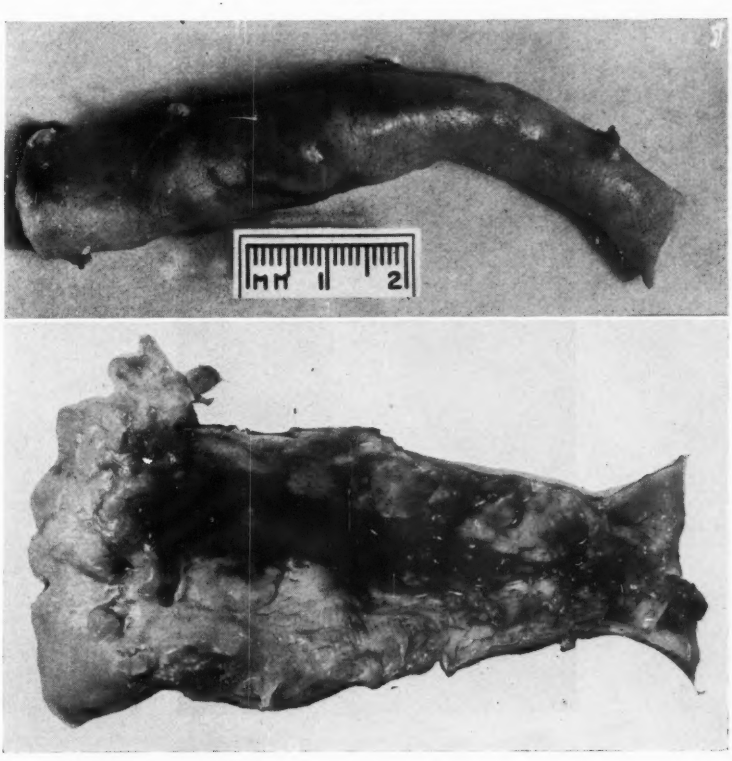
Roentgenogram of the chest revealed erosion of the inferior aspect of the right ninth rib. Scattered areas of calcification were present anterior to the lower thoracic and upper

TABLE IB. RESECTIONS OF THE THORACIC AORTA WITH REPLACEMENT BY HOMOGRAFT FOR CONSTRICTIVE LESIONS

Case	Lesion	Etiology	Date of Operation	Method of Repair	Length of Graft	Period of Occlusion	Result
1. R.A. 46yr. W♂		Syphilitic	1-5-53		15 cm.	45 min.	Excellent
2. O.J. 50yr. N♂		Syphilitic	6-11-53		23cm.	45 min.	Excellent Slight weakness in legs.
3. T.M. 58yr. W♂		Syphilitic	11-9-53		30cm.	77 min.	Died 12 hours later Shock
4. E.McC. 50yr. N♀		Syphilitic	11-19-53		12 cm.	38 min.	Excellent
5. D.J. 17yr. W♀		Congenital (Recurrent)	12-25-53		7 cm.	53 min. hypotherm.	No neurologic sequelae Septicemia Died 8days later.
6. W.McC. 31yr. W♂		Traumatic (Recurrent)	2-5454		8cm.	62 min. hypotherm.	Excellent No neurologic sequelae
7. J.B. 66yr. N♂		Syphilitic	3-4-54		18cm.	53 min. spinal-hypotherm.	Died 6hours later Shock.

lumbar vertebral bodies, suggesting calcification of the aorta. The arch of the aorta was normal in appearance, and the site of coarctation was not visualized. An aortogram done in another hospital was unsatisfactory.

On June 16, 1953, a left posterolateral thoracotomy was done, entering the chest through the bed of the resected fifth rib. The collateral vessels in the chest wall were not as well developed as would be expected in the usual coarctation. The arch and proximal descending aorta were normal. Beginning in the lower third of the descending thoracic aorta there was a constriction and almost complete occlusion of the aorta, ex-



A.

B.

Fig. 19.—Photograph of specimen in Case 8 showing (*A*) the irregular cone-shaped configuration of the constricted segment, and, in opened specimen (*B*), the ragged atherosclerotic lumen with almost complete obstruction at the lower end.

tending over a distance of approximately 8 cm. (Table IB). The constriction was surrounded by a zone of fibrosis, scarring, and multiple small collateral vessels. The sclerotic process appeared to end at the aortic hiatus where the aorta was again soft and of normal texture. In order to gain proper access to the lesion in the lower aorta, it was necessary to resect a segment of the eighth rib and enter the pleural cavity at that level. The constricted segment of aorta was then excised between clamps and the resulting defect was bridged with a segment of aortic homograft preserved in modified Tyrode's solution measuring 9 cm. in length.

His recovery was complicated by emotional instability and mental confusion. Upon discharge from hospital on June 28, 1953, there were pulses in the femoral arteries which

were palpable but not very forceful. Blood pressure in the arms was 180/110 mm. Hg and in the legs was 160/120.

The specimen measured about 9 cm. in length and contained heavily calcified atherosclerotic plaques (Fig. 19). Near the distal end the orifice was about 3 mm. in diameter. The tissue at the margins of the specimen appeared normal in texture and the arteriosclerotic process ended abruptly. During the ensuing months which he spent at home, the emotional instability and mental confusion referable to the previous stroke improved somewhat. On Oct. 20, 1953, he was readmitted to the hospital. Blood pressure in the left arm was 160/110 and in the left leg 110/90. Pulses in the legs were weak.

A.

Fig. 20.—*A* and *B*, Aortogram and explanatory diagram in Case 8 made four months after first operation showing graft in place and second constriction involving the proximal abdominal aorta.

Retrograde aortography revealed a constriction in the proximal portion of the abdominal aorta just above the superior mesenteric and renal arteries and occluding the celiac axis (Fig. 20). The previously inserted thoracic homograft revealed no evidence of dilatation.

On Oct. 26, 1953, a left posterolateral thoracotomy was done through the ninth interspace. The diaphragm was detached posteriorly, and the left crus of the diaphragm and the aortic hiatus were opened. In the proximal abdominal aorta there was an occlusion similar to the one previously resected in the terminal thoracic aorta (Table IB). The abdominal occlusion, which was arteriosclerotic and calcified, measured about 2.5 cm. in length and obliterated the origin of the celiac axis which was represented by a fibrous cord (Table IB).

The previous graft was exposed and its terminal portion was mobilized and excised along with the segment containing the constriction. A lyophilized homograft, approximately 5 cm. in length, was inserted to bridge the defect between the graft in the thoracic aorta proximally and the abdominal aorta distally (Fig. 21). The graft, which had been in place for four months, was in good condition.

His postoperative convalescence was smoother than after the first operation, and he was discharged from the hospital in good condition on Oct. 26, 1953.

When last examined on Feb. 15, 1954, blood pressure in the right arm was 140/90 mm. Hg, right leg 170/100. Pulses in the lower extremities were full and bounding, and he was entirely free of headaches.

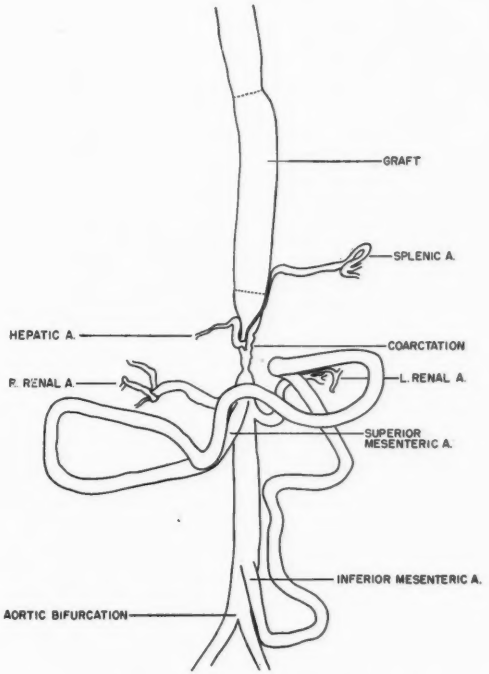


Fig. 20, *B.* *(For legend see opposite page.)*

Comment.—This case resembles others which have been previously reported in which the thoracic aorta in young patients is obliterated by an arteriosclerotic process confined to a localized segment of aorta usually in the middle and distal thirds. It is unique in that there was a short "skip area" at the aortic hiatus in the diaphragm where the aorta was uninvolved, although there was another constriction involving the proximal abdominal aorta. The celiac axis was chronically obliterated by this latter process and gradual occlusion of this vessel had produced no ill effects.

The second operation provided an opportunity for examination of the thoracic graft which had been inserted four months previously. After the

terminal end of the graft was mobilized, it was occluded and the lower end was amputated for histologic study and will be reported elsewhere.[25] Grossly the graft was supple and elastic, and the intimal surface was smooth and glistening. Anastomosis to the lower end of the graft was satisfactory, and the sutures had no tendency to tear out. Because of the patient's religious convictions, no blood transfusions were given during either operation, and blood substitutes were unnecessary.

CASE 9.—S. S., a white boy, aged 12 years, was admitted to Methodist Hospital on Aug. 13, 1954, with mild cramps in his legs usually occurring at night. Several months prior to admission, while swimming, he developed severe precordial pain lasting several minutes. Except for these symptoms his course and development were uncomplicated.

Physical examination revealed a well-developed healthy boy. Blood pressure in the right arm was 155/90 mm. Hg, left arm 110/90, and in the legs was unobtainable. Right brachial and both carotid pulses were normal, but no pulses were palpable in the left arm or either leg. There was a systolic murmur best heard over the base of the heart and transmitted upward into the vessels of the neck. A bruit was audible in the interscapular region, and there were visible and palpable collateral arteries in the superficial muscles of the back.

Roentgenograms of the chest revealed in the left upper mediastinum in the position usually represented by an aortic knob a lobulated mass which under fluoroscopy did not pulsate (Fig. 22). A retrograde aortogram revealed a narrowing of the aorta distal to the left common carotid artery, and beyond this area was a lobulated aneurysmal mass filling completely with contrast medium (Fig. 23).

On Aug. 17, 1953, exploratory left posterolateral thoracotomy was performed, and the coarctation and aneurysm were exposed (Table IB). The sac was exceedingly thin-walled on its lateral surface and the left subclavian artery originated on the posterior surface of the aneurysm about 4 cm. distal to the coarctation (Fig. 24). The aorta was clamped proximal to the coarctation and distal to the aneurysm, and the subclavian artery was occluded separately. The abnormal segment of aorta was excised and a segment of lyophilized homograft of aortic arch was used to bridge the defect restoring not only aortic but also subclavian arterial continuity (Fig. 25). The aortic anastomoses were completed first and while blood was circulating through the aortic portion of the graft, the subclavian anastomosis was completed.

His postoperative course was uncomplicated, and he was discharged from the hospital eight days later. When last examined six months later, there were excellent pulses in all extremities. Blood pressure in the right arm was 120/60 mm. Hg, left arm 100/60, left leg 130/70.

Comment.—Aortic reconstruction in this case was very satisfactory in that not only was aortic continuity restored but also normal circulation to the left subclavian artery was obtained. Therefore, no major vascular tributary was unnecessarily interrupted.

CASE 10.—A. T., a 20-year-old white man, was admitted to the Methodist Hospital on Sept. 13, 1953, with a diagnosis of coarctation of the aorta. At the age of 17, hypertension was discovered but he had experienced no symptoms referable to the lesion.

Physical examination revealed an extremely well-developed, muscular individual in apparent excellent health. Blood pressure in both arms was 190/110 mm. Hg, and in the lower extremities the blood pressure and pulses were unobtainable. A soft systolic murmur was present over the base of the heart and posteriorly a bruit was audible in the interscapular region. Large collateral vessels were palpable in the chest wall.

Fig. 21.—Photograph made at second operation in Case 8 showing homograft in place. Proximally, the graft is sutured to lower end of another graft inserted four months previously, and, distally, graft is sutured to abdominal aorta just above superior mesenteric artery.

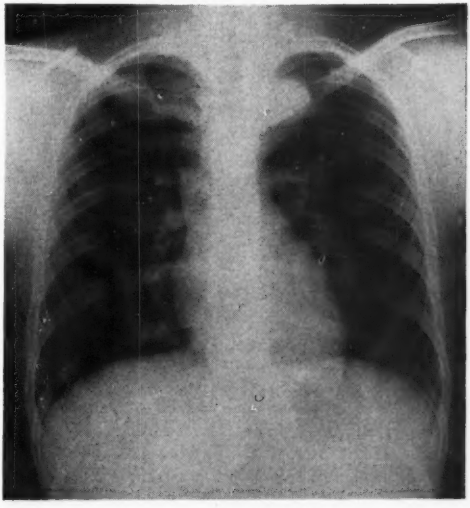


Fig. 22.—Roentgenogram of chest in Case 9 showing lobulated mass in left superior mediastinum.

Roentgenograms of the chest revealed erosion of the inferior margins of many of the ribs on both sides, and the aortic knob was small.

On Sept. 15, 1953, a left posterolateral thoracotomy was performed, and a long narrow coarctation distal to the level of the left subclavian artery was demonstrated measuring 3 to 4 cm. in length and originating distal to the left subclavian artery (Table IB). There was an aneurysm about 3 cm. in diameter near the site of coarctation and three other 0.5 cm. aneurysms associated with intercostal and bronchial arteries. The segment containing the aneurysms was resected, and the 4 cm. aortic defect was bridged with a lyophilized aortic homograft.

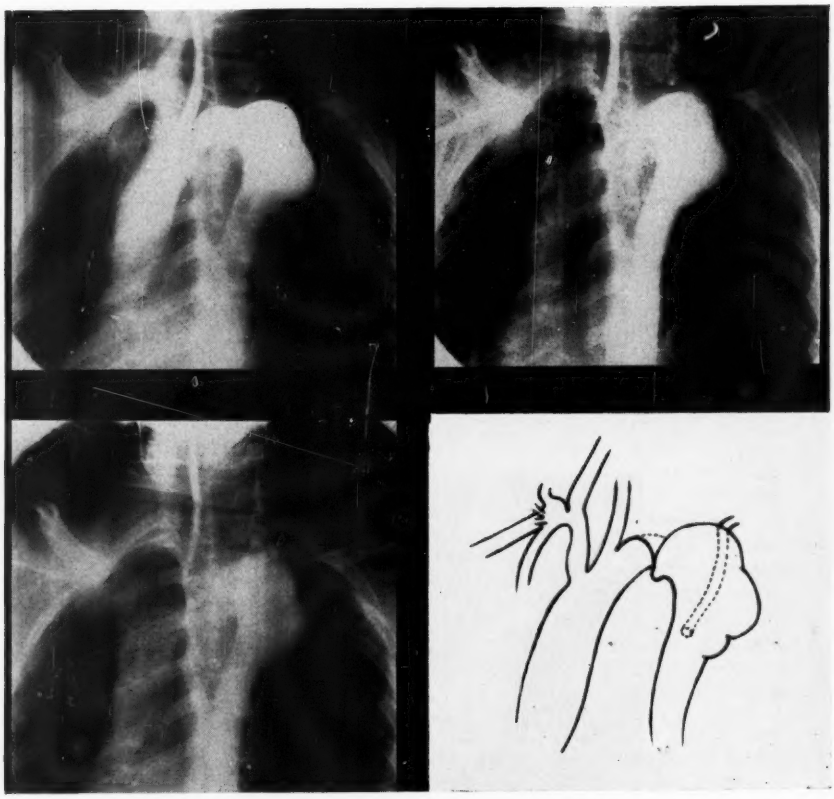


Fig. 23.—Retrograde aortogram in Case 9 showing coarctation of aorta with lobulated aneurysm distal to the constriction and anomalous origin of left subclavian artery.

His postoperative progress was smooth, and he was discharged from the hospital in excellent condition seven days later. When last examined on Jan. 14, 1954, the blood pressure in the right arm was 140/90 mm. Hg, right leg 170/100 mm. Hg, and roentgenogram of the chest showed a normal configuration of the superior mediastinum.

CASE 11.—J. H., a 30-year-old white male carpenter, was admitted to Methodist Hospital on Oct. 25, 1953. A diagnosis of coarctation of the aorta was made in 1945 while the patient was on active duty with the Navy, but he had experienced no symptoms until the recent sudden onset of severe headache associated with vomiting and blurring of vision.

During the week prior to admission he had noticed tingling sensations in the right hand, moderate stiffness of the neck, and mild tinnitus. Intracranial hemorrhage was strongly suspected.

Physical examination revealed forceful pulsations in the suprasternal notch. The right radial pulse and both carotid pulses were normal, but the left radial pulse as well as pulses in the legs were weak. Blood pressure in the right arm was 190/110 mm. Hg, left arm 140/130, and in the legs was unobtainable. The heart was not enlarged, and there was a Grade II aortic systolic murmur audible to the right of the sternum in the second interspace. Collateral vessels palpable in the chest were much more striking on the right, and a pronounced systolic bruit was present to the right of the midline in the interscapular region.

Fig. 24.

Fig. 25.

Fig. 24.—Photograph made at operation in Case 9 showing lobulated aneurysm of aorta with descending thoracic aorta extending to left. Vessel originating on posterior aspect of aneurysm and directed toward right in photograph is anomalous left subclavian artery.

Fig. 25.—Photograph made at operation in Case 9 showing aortic arch homograft in place after resection of coarctation and aneurysm. Circulation in left subclavian artery has been restored by anastomosis with corresponding vessel on graft.

Roentgenograms of the chest revealed the heart to be of normal size with absence of the normal aortic knob (Fig. 26). There was deep notching of the undersurfaces of the ribs most pronounced on the right side.

Left thoracotomy was performed on Oct. 27, 1953, and a coarctation of the aorta 3 cm. long commencing just distal to the left common carotid artery was identified (Table III). There were two large vessels of almost equal size originating in the constricted zone, one of which was probably an anomalous vertebral artery and the other the left subclavian artery. The anomalous segment of aorta was resected, and a lyophilized aortic homograft was inserted in the defect. A graft of aortic arch was utilized including the left common carotid and left subclavian arteries and by this means it was possible not only to restore

aortic continuity but also to connect the two large arterial trunks to the graft (Fig. 27). Upon completion of the aortic anastomoses, aortic circulation was permitted through the graft while the anastomoses of the lesser vessels were done.

His postoperative recovery was uneventful, and he was discharged from the hospital nine days later. He was examined five months after operation, at which time he was asymptomatic and was again working as a carpenter. Pulses in all extremities were strong and the blood pressure in the right arm was 150/90 mm. Hg, left arm 120/90, and left leg 130/100.

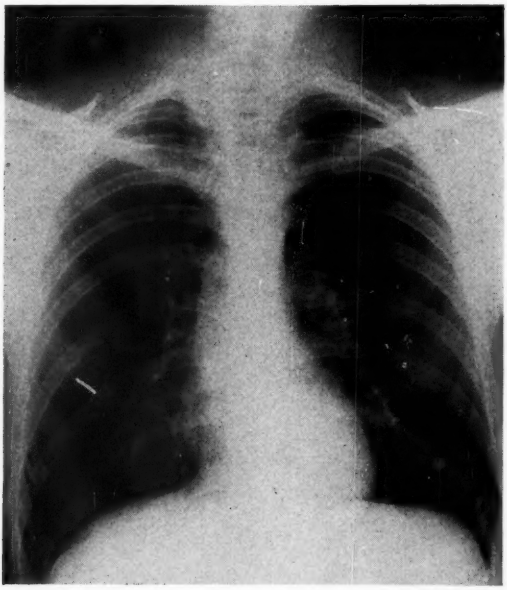


Fig. 26.—Roentgenogram of chest made in Case 11 showing absence of aortic knob in superior mediastinum and erosion of undersurfaces of ribs most striking in right hemothorax.

Comment.—This case is somewhat similar to another which was previously reported by Blalock[7] in which the subclavian artery was divided and end-to-end anastomosis was possible. In our case the length of the coarctation precluded end-to-end anastomosis. The method of repair used in our case provided an ideal functional result.

CASE 12.—J. O'G., a 29-year-old white woman, was admitted to Methodist Hospital on March 25, 1954, with a history of hypertension for the past five years. She had been asymptomatic except for occasional pounding sensations in the blood vessels of the neck. Physical examination revealed blood pressure of 160/90 in the right arm, 160/110 in the left arm, and 0/0 in the right leg. The pulses in the arms were equal bilaterally, but no pulses were palpable in the lower extremities. The aortic second sound was markedly accentuated, and a systolic bruit was heard in the scapular region posteriorly. Collateral arterial pulsations were palpable in the chest wall.

Roentgenograms of the chest revealed mild cardiac enlargement with absence of the cardiac knob and notching of the undersurface of the ribs bilaterally.

On March 26, 1954, at thoracotomy the coarctation was demonstrated to begin just distal to the left subclavian artery and extend for a distance of approximately 3.5 cm.

with relatively little poststenotic dilatation of the aorta (Table IB). The coarctation was excised, but because of the extent of the defect a 4 cm. lyophilized homograft was used to repair the defect.

Postoperative convalescence was uneventful, and she was discharged from the hospital eight days later. One month later the blood pressure in the right arm was 150/90 mm. Hg, left arm 146/90, right leg 150/110.

Fig. 27.—Photograph made at operation in Case 11 showing on left the elongated segment of coarctation and two large arteries, presumably the left vertebral and subclavian arteries, arising in constricted zone. Photograph in inset shows aortic arch homograft in place and left vertebral and subclavian arteries anastomosed to stumps of left common carotid and subclavian arteries on graft, respectively.

SUMMARY

For fusiform aneurysms and constrictive lesions of the descending thoracic aorta, resection of an extensive segment of aorta may be necessary making end-to-end anastomosis impossible. In such cases restoration of aortic circulation may be achieved by insertion of vascular homografts. This method of therapy was employed in twelve thoracic aortic resections, seven of which were done for aneurysm and five for constrictive lesions.

The degree of ischemic damage to the spinal cord occurring during periods of temporary aortic occlusion is influenced by a number of factors, including the duration of occlusion, extent and location of the lesion, and extent to which collateral circulation exists. In constrictive lesions the collateral circulation is usually well developed, and temporary aortic occlusion may be tolerated for indefinite periods. In aneurysms, however, where collateral circulation is poorly developed, successful performance of aortic resection depends upon prevention of ischemic damage to the spinal cord during temporary

aortic cross-clamping. Two cases of aneurysm are reported in which general body hypothermia was employed, and another in which local spinal cooling was used to prevent postoperative paraplegia. Other means of reducing this hazard are concerned with the technical procedure itself.

In constrictive lesions, where there is no haste in restoration of aortic flow, emphasis is placed upon precise reconstruction of all major vascular channels by homograft. One case of acquired coarctation of the aorta is reported in which constrictions occurred in the lower thoracic aorta and in the proximal abdominal aorta. Good results were obtained after two homografts were inserted to replace the diseased segment of aorta.

Addendum.—Since the preparation of this manuscript, 9 additional cases have been performed, 5 of which were done for aneurysms and 4 for constrictive lesions. All of these patients recovered, making a total of 21 cases with 3 deaths (14.3 per cent). Of particular interest is the fact that two of the additional patients had aneurysms involving the distal part of the aortic arch similar to that described in Case 6. In both of these patients the resection and graft replacement was done under general hypothermia as described in Case 6. The proximal occluding clamp was applied just distal to the left common carotid artery in each for periods of 58 and 50 minutes, respectively. Both have completely recovered with no evidence of ischemic damage to the spinal cord or other viscera.

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Discussion

(Papers by Stranahan, Alley, Sewell, and Kansel; and Cooley and DeBakey)

DR. CONRAD R. LAM, Detroit, Mich.—I wish to compliment the essayists of the last two papers for their very excellent application of these rather new surgical principles. It certainly requires a great deal of patience and courage. This operative work also requires a supply of homografts, and I should like to mention a method we have used for graft sterilization.

We have used a chemical named betapropiolactone* for sterilization of plasma and blood. Observation of such a sterilized graft implanted in an animal for two months shows that the graft has a perfectly normal appearance. We have observed other grafts over a period of two years. The hemotoxylin and eosin stain shows the graft almost indistinguishable from the host. With trichrome stain, we see the graft side with the intimal thickening and, on the host side, we see the more normal media without intimal thickening.

In a greatly enlarged view of the normal for comparison, one notes that the muscular tissue has disappeared, leaving only the elastic tissue. This is quite typical of a perfectly functioning homologous graft.

My general surgical colleague, Dr. Szilagyi, has used sixteen of these sterilized grafts in human cases. He has replaced the bifurcation of the aorta four times, the common iliac twice, and the femoral artery ten times. All of these grafts have appeared to serve their purpose well. The longest follow-up period is six months.

DR. CHARLES P. BAILEY, Philadelphia, Pa.—I would like to present a case which may open up another field for surgery of aneurysms. Recently we operated on a patient with a large ventricular aneurysm which extended apparently from the apex of the left ventricle to the bifurcation of the left coronary artery. The anterior descending branch was obliterated by the occlusion, producing the infarction which was the cause of this aneurysm. As you know, ventricular aneurysms nearly always are caused by infarction and are said to occur in about 15 per cent of all acute coronary occlusions. Because pathologic study of these lesions indicates that these aneurysms have very tough fibrotic bases, and since they rupture at the fundus rather than at the base, we thought it would be safe to put a clamp across the base of the aneurysm, cut it off, and oversew it. The largest clamp we had, the double length Satinsky clamp, only extended across about two-thirds of the aneurysm, so we placed large sutures in continuous mattress fashion, through the base of the sac from one extremity of the ventricle to the other. Then by traction on the extremities of the sutures, the loops became straightened out and pulled the entire base together in a straight line. Then we placed another row of interrupted mattress sutures. Finally the sac was amputated and the cut edges were oversewn with a third row of sutures, which we thought would be good for reinforcement. There were clots in this region, although near the apex of the ventricle there were none.

Actually, this is not the first such operation. Dr. Robert Glover performed a similar operation almost one year ago, and I am sure it would have been successful except that the patient bled to death from a gastric ulcer several days after surgery. It appeared good at autopsy. There may have been other similar cases of which I am not aware. Of course we know about the work of Drs. Murray, Allen, and Beal in the experimental resection of infarcts, which was fundamental to this concept. They were attempting to operate on acute infarctions of the heart, however, which I think would be much more risky.

This patient, who preoperatively had heart failure, shortness of breath, and gallop rhythm that could not be relieved medically, on the operating table developed a higher blood pressure and better cardiac action as soon as this paradoxically dilating aneurysm was removed. He has had no gallop rhythm and no dyspnea since the operation. I think that this technique might be applied rather generally in those patients who develop aneu-

*Obtained from the B. F. Goodrich Research Laboratories, Brecksville, Ohio.

rysms following myocardial infarction, if they show evidence radiographically of progressive enlargement and impending rupture, or present severe disability. The pathology would seem to indicate that the sutures will hold well, and this clinical experience suggests that they may become greatly improved.

DR. HENRY T. BAHNSON, Baltimore, Maryland.—I enjoyed both these presentations and have only one point I would like to make, that is, that a graft is not always required. Syphilis and trauma usually cause localized aneurysms, and frequently one may perform an aortorrhaphy without the use of a graft. We have required a shunt and a graft in only one of the sixteen thoracic aneurysms that we have treated surgically; this one was treated unsuccessfully, however. Certainly the contributions which have been made in the form of shunts and hypothermia will add to the safety of inserting grafts. I think the principle of a lateral aortorrhaphy is valid, particularly with traumatic aneurysms, because in order for a patient to survive a traumatic aneurysm the tear almost has to be a fairly limited one. The two shown here occurred in the typical position for a traumatic aneurysm. The common cause at present is an automobile accident, and not always with the split pea and walnut syndrome.

DR. JAMES FINNERTY, New York, N. Y.—I would like to make a comment about the importance of maintaining collateral circulation in coaretation of the aorta. Two years ago we had the unfortunate experience of resecting the coaretation in a patient who had an innominate artery that originated from the distal aorta immediately beyond the coaretation and, not having recognized this anomaly preoperatively, in order to gain strength, we divided the innominate artery. This patient postoperatively had a transient left hemiplegia, absent pulse in the right arm, and absent right carotid pulse. The left hemiplegia cleared, but the patient was left with persistent paraplegia. I think if we bear this in mind we might occasionally avoid this complication.

One aneurysm of the ascending aorta was present in a rather unusual position, namely, on the left side. The angiogram failed to show increased opacification of the aneurysm. At operation it was possible to do a lateral aneurysmorrhaphy, as suggested by Dr. Bahnson, and the patient has done well since.

LT. WILLIAM H. SEWELL (Closing).—The decision to use a heterograft in the first case was based largely on some work done at the Naval Medical Research Institute at Bethesda.

Studies were carried out in which aqueous extracts were made of both fresh and freeze-dried arteries. Rabbits were immunized with these extracts, and later were challenged with the same antigens. All the rabbits that were immunized with the fresh extract and challenged with the same one showed a positive precipitin test; those that were immunized with the extract of the freeze-dried artery and challenged with that showed no precipitins. The ones in which one extract was used as immunizing antigen and the other for the challenge, and vice versa, showed a positive precipitin in some of the animals.

We concluded that these findings may well explain the fact that there was less foreign body reaction in the freeze-dried grafts than there was in the fresh in our series. There was less fibrosis around the freeze-dried grafts. They lost elasticity more rapidly, but soon leveled off, whereas the fresh grafts continued to lose it for a long period of time, and ended up much less elastic.

In the freeze-dried grafts the muscle degeneration occurs much more rapidly than in the fresh, and replacement by fibroblasts also occurs more rapidly and is more nearly complete.

The difference between the fresh and freeze-dried homografts was not very great. The freeze-dried heterografts, however, transplanted from the pig to the abdominal aorta of the dog, showed that the results were almost as good grossly as those of the fresh homograft. This is certainly a much better result than would be obtained if we simply took a fresh heterograft from a pig and put it into the dog. We feel that heterografts are not preferable to homografts, and the reason it was used was because the man's aorta above

and below the aneurysm was so dilated that it was impossible to fit it with a homograft. The graft was taken from two calves, and even so it was smaller than the aorta, but it was a much closer fit than could have been obtained with a human vessel. The calf aorta is two to three times the diameter of the human aorta.

I think that plastic cloth would be satisfactory for most types of aortic grafting, but in an area where there are large branches coming off, I suspect that the turbulence which would occur would give propagation of the thrombus lining the inside of the cloth and probably would cause trouble.

DR. M. E. DEBAKEY (Closing).—I should like to express our appreciation to the men who have discussed this paper. With reference to a point made by Dr. Bahnson regarding lateral aortorrhaphy, we too are convinced that it is often the procedure of choice. That is particularly true in syphilitic aneurysms where there is a relatively small sacciform type of aneurysm. This procedure, as Dr. Bahnson and those interested in it know quite well, was first developed by Dr. Tuffier more than fifty years ago, and the principles of the operation were clearly described by him, although he himself was never successful in applying it.

So far as heterologous grafts are concerned, our experimental observations lead us to feel that they are not as satisfactory as homografts, to the extent that we would be very hesitant to use a heterograft in a human patient. So far as the procedure of shunts is concerned, we realize that this may have definite application in the resection of the arch where the carotids and innominate artery may have to be occluded for a significant period of time. The success we had with the first patient, however, on whom we reported about a year ago, in resecting an aneurysm of the descending thoracic aorta without the use of a shunt, made us feel that to introduce this procedure would complicate the operation if it was not absolutely essential. For this reason we are inclined to feel that in the resection of aneurysms involving the descending thoracic aorta up to the level of the subclavian, with the use of hypothermia of moderate degree, and the expedition of the operation so that the occlusion time is not beyond an hour, it is possible to do this operation quite successfully without the use of shunts and without complications.

Finally, one point I would like to make is with regard to the ultimate fate of homografts. Dr. Gross, as you know, has had an extensive experience with the use of grafts for a longer period, perhaps, than anyone else, and has found that they have stood up well, certainly for five or six years. We have had the fortunate opportunity to excise pieces of graft after varying periods up to one year, and the study of these grafts has shown that they behave very much as experimental observations would indicate that they would behave, and at the end of one year their architectural structure is still functionally satisfactory. For this reason we are inclined to believe they will provide satisfactory results for a much longer time.

THE ARTERIAL OXYGEN TENSION AFTER RESECTION WITH SIMULTANEOUS OSTEOPLASTIC THORACOPLASTY

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THE aim of treatment for pulmonary tuberculosis is to achieve persistent negative guinea pig tests of the sputum and gastric washings with a maximum preservation of pulmonary function. To achieve this result, a combination of antimicrobial therapy, bed rest, resection, and thoracoplasty must be used. The treatment must be individualized for the cases with pulmonary tuberculosis. In a large group of cases, however, it is obvious that all four therapeutic measures must be utilized. In this group we consider a resection with simultaneous osteoplastic thoracoplasty the treatment of choice.

A preliminary thoracoplasty with secondary resection has several disadvantages:

1. When the patient recovers from the first operation he has to look forward to a second major one.
2. The danger of spread is greater after a thoracoplasty than after a resection.
3. The hospitalization of the patient is prolonged from three to six months.
4. It is more difficult to model the chest in the desired size at a second operation.

The only advantage with the two-stage procedure was that there was no paradoxical movement at the second operation. The resection was therefore considered safer when the thoracoplasty had already been performed.

The new osteoplastic thoracoplasty, according to Björk,[1-2] can be made in any desired size and will make a stable roof for the thorax when the resection is completed. As the paradoxical movements are excluded, the patients will stand this combined procedure very well. In the first one hundred cases of resections with simultaneous osteoplastic thoracoplasty, only one was lost.[2] The operation is only prolonged by thirty to forty-five minutes. The patients need, however, three or four extra blood transfusions, as usually 1,500 to 2,000 ml. of bloody fluid is drained off during the first forty-eight postoperative hours. In this paper we intend to show the arterial oxygen tension in the postoperative course after resections with simultaneous osteoplastic thoracoplasty as compared to resection under a preliminary thoracoplasty.

MATERIAL

Thirty consecutive patients with pulmonary tuberculosis undergoing resection with simultaneous osteoplastic thoracoplasty were investigated (Group

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This work was aided by The Swedish National Association against Tuberculosis.
Received for publication May 17, 1954.

I). For comparison, twenty-nine patients with pulmonary tuberculosis with a preliminary thoracoplasty were investigated when undergoing resection at a second stage (Group II).

METHOD

With an indwelling needle in the femoral artery, a blood sample was taken at rest after the patient had been hyperventilating as much as possible for about twenty-five seconds. Oxygen and carbon dioxide tensions (pO_2 and pCO_2) in the arterial blood were determined according to Riley, Proemmel, and Franke[7] with a slight modification. The saline or saline-Dreft solution has been omitted for washing the capillary. The sodium hydroxide solution is applied for both the capillary washing and the simultaneous carbon dioxide combining, thereby eliminating one source of error.[3] This investigation was performed before operation and at intervals during the postoperative period.

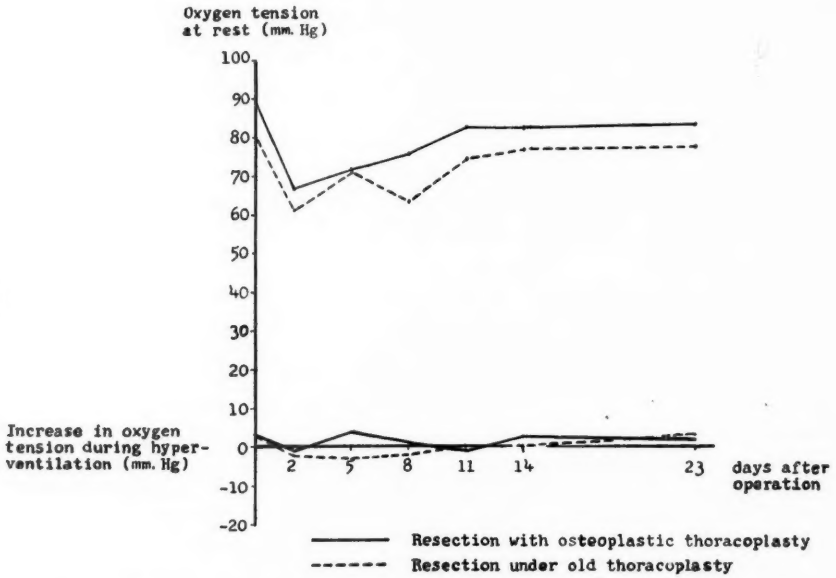

Fig. 1.—The two curves for resting arterial oxygen tension are, on the whole, parallel whether the resection is done with simultaneous osteoplastic thoracoplasty or under an old thoracoplasty.

RESULTS

The investigation has shown a decrease of the resting arterial oxygen tension during the postoperative course of the same magnitude in the two groups (Table I, Table II, and Fig. 1). Thus the two curves for resting arterial oxygen tension are, on the whole, parallel, whether the resection is done with simultaneous osteoplastic thoracoplasty or under an old thoracoplasty. Nor

has it been possible to see any significant difference in the two groups in the arterial oxygen tension during voluntary hyperventilation (Table I, Table II, and Fig. 1).

DISCUSSION

The stable roof of the thorax obtained by the osteoplastic thoracoplasty will exclude paradoxical respiration and facilitate an effective cough. Thus no more cases of atelectasis are found if this thoracoplasty is added at the same time as a resection, as compared to the group of resections under primary thoracoplasties.

The investigation of the resting arterial oxygen tension during the postoperative period has confirmed the clinical impression that the osteoplastic thoracoplasty is a physiologic operation that can be added to a resection when indicated without hesitation from a functional point of view.

TABLE I. RESECTION WITH OSTEOPLASTIC THORACOPLASTY

	BEFORE OPERATION	DAYS AFTER OPERATION					
		1-3	4-6	7-9	10-12	13-15	16-30
Resting Oxygen Tension (mm. Hg.)							
Number of cases	30	15	14	15	11	19	8
Mean value	89.1 ± 1.0	66.8 ± 2.3	71.9 ± 2.1	75.9 ± 1.9	82.4 ± 1.3	82.1 ± 1.4	82.7 ± 2.0
Standard deviation	5.6	8.9	7.8	7.5	4.3	6.0	5.8
Increase in Oxygen Tension (mm. Hg) During Hyperventilation							
Number of cases	29	14	13	15	10	19	8
Mean value	3.0 ± 1.3	−1.0 ± 1.7	+3.7 ± 1.9	+1.1 ± 1.6	−0.3 ± 1.7	+2.8 ± 1.2	+2.0 ± 3.1
Standard deviation	6.7	6.5	6.7	6.2	5.4	5.3	8.8

TABLE II. RESECTION UNDER OLD THORACOPLASTY

	BEFORE OPERATION	DAYS AFTER OPERATION					
		1-3	4-6	7-9	10-12	13-15	16-30
Resting Oxygen Tension (mm. Hg)							
Number of cases	31	6	5	7	4	17	6
Mean value	80.2 ± 1.9	61.4 ± 5.2	70.7 ± 3.1	63.5 ± 3.0	74.2 ± 1.0	76.3 ± 2.2	76.0 ± 3.9
Standard deviation	10.5	12.8	6.9	8.0	1.9	8.9	9.5
Increase in Oxygen Tension (mm. Hg) During Hyperventilation							
Number of cases	31	6	5	7	4	17	6
Mean value	2.8 ± 1.5	−1.4 ± 1.7	−3.3 ± 3.2	−2.2 ± 2.4	+0.2 ± 2.3	+0.3 ± 2.3	+2.1 ± 1.3
Standard deviation	8.2	4.3	7.2	6.3	4.6	9.6	3.1

A patient's ability to increase the arterial oxygen tension during voluntary hyperventilation in the immediate postoperative period will partially depend on the pain. The increase of oxygen tension during hyperventilation in patients undergoing resection with osteoplastic thoracoplasty has not been less than in cases with resection under old thoracoplasties. We believe this confirms the clinical impression that the patients with the combined operation have no more pain than patients with the two-stage procedure.

SUMMARY

The arterial oxygen tension at rest and during voluntary hyperventilation has been investigated during the early postoperative course in thirty pa-

tients undergoing resection with simultaneous osteoplastic thoracoplasty. The results have shown this to be a well-tolerated procedure and that there is no reason from a functional point of view to perform resection with thoracoplasty in two stages.

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